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American Heart Journal

VOL. 50 DECEMBER, 1955 NO. 6

Original Communications

PHONOCARDIOGRAPHIC STUDY OF PULMONIC-SYSTOLIC MURMURS IN CHILDREN

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THE problems of cardiac evaluation and diagnosis in children and young adults are considerably complicated by the frequency of occurrence of nonpathologic cardiac murmurs among subjects of these age-groups. In various large series of subjects studied, the rate of occurrence of such nonpathologic murmurs has usually been reported in the range of 40 to 60 per cent,[1-6] although in other series per cents of 12 to 20 have been reported.[7-10] The differentiation of such murmurs from those of pathologic significance may involve considerable uncertainty. Although useful generalizations have been made, it is generally accepted that no combination of the criteria or measurements employed in physical examination can serve to differentiate all of these "functional" cardiac murmurs from the organic ones.[6,11]

In the present studies it has been found useful to consider these nonpathologic murmurs of children in two groups, since these groups embrace at least 95 per cent of all such murmurs encountered. Such grouping of functional murmurs is recognized in other clinics as well. The first of these is the precordial murmur described by Still[12] early in the century as the "twanging-string murmur." It is of a buzzing quality, usually not at all harsh, and is generally of maximal intensity in the midprecordium. This murmur decreases in intensity in adolescence and is virtually never heard in adult subjects. The other, the pulmonic-systolic murmur, is of maximal intensity in the pulmonic area. (The name derives from this feature, not from any implied association with the pulmonary artery.) It may be heard with diminishing intensity across the sternum, or down the left border of the sternum, or along a diagonal line toward the apex. It can be heard in a range of degrees of roughness or harsh-

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ness in different subjects. With increasing age of the subject, the pulmonic-systolic murmur generally decreases in intensity and in its distance of "transmission," so that in those subjects in whom the murmur persists into adult life it is generally of lower intensity and usually confined to the pulmonic area. Murmurs which appear to be of nonpathologic significance are occasionally found outside of these two groups, e.g., with points of maximum intensity to the right of the sternum, or at the lower left border of the sternum or at the apex, but these are quite rare in comparison with the total of the two categories described previously. Within these two categories there are not infrequently instances of overlapping, in which the application of the criteria mentioned previously leaves possible differences of judgment as to the category involved. However, in general the classification of the large majority of all functional murmurs into these two groups is quite clear.

The distribution of functional murmurs between these groups is stated or implied in some of the studies referred to here. The relative percentages thus found have varied among these reports, perhaps to some extent on the basis of the age-group under study. Thus Friedman and associates[5] found 39 per cent of the 234 functional murmurs in a series of 500 children of 2 to 12 years of age to be pulmonic-systolic murmurs, and Messeloff[11] found 22 per cent of 500 functional murmurs in school-age children to be of maximal intensity in the second intercostal space to the left of the sternum. (Of the remaining functional murmurs a considerable number were of maximal intensity in the third intercostal space to the left of the sternum, and some of these may have been pulmonic-systolic rather than precordial "twanging-string" murmurs.) Among older subjects Schwartzman,[3] Contratto,[8] and Stewart[6] found that of their series of functional murmurs 53, 56, and 60 per cent were referable to the base of the heart in respective series of high school students, college students, and unspecified adults.

Comparative studies of the precordial "twanging-string" functional murmur and of murmurs of valvular insufficiency by phonocardiography, with especial reference to wave form of the murmurs, were carried out in this laboratory recently.[13,14] In these studies apparently characteristic differences in wave form were noted between the phonocardiograms of the two types of murmurs. The wave form seen in the phonocardiogram of the precordial "twanging-string" murmur was that of a simple, uniform wave of constant frequency, resembling in general a damped sine wave of a frequency in the range of 70 to 130 cycles per second, whereas that of the murmurs of valvular insufficiency were so complex that no pattern or fundamental frequency could be discerned. Because of the observation of this apparently characteristic difference between murmurs of valvular insufficiency and one of the two large groups of nonpathologic murmurs, the present study was undertaken of the other large group of functional murmurs, the pulmonic-systolic murmurs.

METHODS AND MATERIALS

Clinical Material.—The patients included in this study were seen in the Rheumatic Fever Clinic of the Philadelphia General Hospital, which functions

in part as a diagnostic clinic and therefore admits many children with miscellaneous undiagnosed cardiac murmurs. These children were examined on their initial visit by the usual clinical tests directed at establishing or ruling out a diagnosis of rheumatic fever or rheumatic heart disease: medical history, physical examination with particular reference to the circulatory system, ECG, roentgenogram of the thorax, and in some cases orthodiagram. Laboratory determinations included the erythrocyte sedimentation rate, the antistreptolysin titer of the serum, and the streptococcal antihyaluronidase titer. (The procedures by which the three last-named tests were carried out have been described elsewhere.[15-17]) Determinations of the red and white blood cell count and of sickling of erythrocytes were done frequently, but not in all cases.

These patients were regarded from the time of the first visit as having no evidence of heart disease, but rather than being discharged from the clinic after a few visits, they were kept on an active status because of this study. After two or three visits had served to confirm the diagnosis, these patients were reexamined at approximately semiannual intervals. They were thus observed, with occasional or periodic repetitions of such tests as seemed to be indicated, for periods ranging from two to ten years, without the development of any evidence of heart disease.

Criteria for Pulmonic-Systolic Murmurs.—The patients selected for this study were those in whom a systolic murmur could be clearly heard over the base of the heart, with a point of maximum intensity in the second intercostal space to the left of the sternum. These murmurs were of varying degrees of harshness and intensity, except that unusually loud murmurs were not included in this study, nor were murmurs which could be heard transmitted to the back. The patients included had no other cardiac murmurs except, in a few cases, precordial vibratory murmurs (the twanging-string murmur of Still[12]), and except for apical systolic murmurs, in the following group mentioned as having rheumatic carditis.

Recording of the Murmurs.—Stethographic tracings were taken at the point of maximum intensity of the murmur (second intercostal space to left of sternum) with the Sanborn Stethocardiette. The medium bell microphone was used for the great majority of the tracings taken in this study. The amplification setting was at a level chosen in an attempt to have the first heart sound produce a wave of 20 mm. in amplitude. The amplitude of the murmur as shown in the tracing is therefore a measure of its intensity relative to the first heart sound. A few tracings were also taken, in the case of some of the murmurs, with the Sanborn Stethocardiette using a logarithmic-response microphone, with the Sanborn Twin-Beam instrument, and with the Cambridge stethograph. These tracings in all cases showed the same general wave form, and the same relation of wave form to that of the precordial vibratory murmur as that described for the Stethocardiette as used with the medium bell microphone.

RESULTS

1. *Duration and Relative Intensity.*—Phonocardiographic recordings were made of the pulmonic-systolic murmurs in forty subjects. For purposes of

comparison tracings were also taken of some clearly identified murmurs of mitral valvular insufficiency and of some precordial vibratory (twanging-string) murmurs. Measurements were made of the duration of the pulmonic-systolic murmurs as recorded, and of their intensity or amplitude in relation to that recorded for the first heart sound. The duration of the murmur ranged from 0.12 to 0.26 sec., with a mean duration of 0.17 sec., and the fraction of the length systole occupied by this murmur ranged from 0.24 to 0.8, with a mean value of 0.46 sec.

2. *Wave Form of the Pulmonic-Systolic Murmur.*—On examination of the physical characteristics of the waves recorded for the pulmonic-systolic murmurs, it was found that certain similarities existed among these and that the pulmonic-systolic murmurs as a group exhibited certain differences from both the precordial vibratory (or twanging-string) murmurs and the murmurs of valvular insufficiency, of which characteristic wave forms have been described previously.[13,14] The wave form of the pulmonic-systolic murmur showed some similarity to that of the precordial vibratory murmur, in that a simple wave could be discerned. However, whereas the form of the precordial vibratory murmur is generally quite uniform, resembling a sine wave of constant frequency, that of the pulmonic-systolic murmur almost invariably showed some distortion, so that there were almost always points in the wave at which the regularity of the simple form was lost. Frequently the regularity of the wave was restored after such distortions, or, at least, the basic simple wave could more easily be discerned in the portions of the wave following the interruption. However, even with these irregularities relative to a simple wave, the wave form of the pulmonic-systolic murmur was quite clearly different from that of the murmur of valvular insufficiency, in which, as in the earlier studies, there was a chaotic mixture of components of far too great a degree of complexity to permit of any visual recognition of a wave pattern.

Traces typical of those found in this study are shown in Fig. 1. In this figure three phonocardiograms showing pulmonic systolic murmurs are presented in the central panels. For comparison, three traces from patients with precordial vibratory (twanging-string) murmurs are shown on the left, and three traces from patients with murmurs of valvular insufficiency are shown on the right. The traces of pulmonic systolic murmurs shown here were selected to illustrate approximately the range of visual degrees of distortion of simple wave form found in these traces. However, even in the traces which show relatively less distortion of the simple wave form, as in the case of the second cardiac cycle of the topmost pulmonic-systolic trace, and the first cycle on the middle one, the departure from the simple wave, as seen on the left, can quite easily be discerned. On the other hand, the difference in complexity of wave form between the group of traces of pulmonic-systolic murmurs and those of the murmurs of valvular insufficiency shown on the right is also quite clear.

3. *Wave-Form of the Pulmonic-Systolic Murmur as a Function of Acoustic Quality.*—Because of the wide range in the degree of harshness or roughness to the human ear which is found among pulmonic-systolic murmurs, these murmurs were graded, at the time of recording, in terms of subjective judgment as to

harshness of the murmurs. The grades noted were as follows: (1) least harsh: similar to, or only slightly rougher than, the buzzing "soft" quality typical of the great majority of precordial vibratory (twanging-string) murmurs; (2) moderately harsh; (3) most harsh: rough quality, approaching the blowing character of murmurs of valvular insufficiency. In the case of some of this last-named group the murmur, if it had been of maximal intensity at the apex, might well have passed for a murmur of mitral insufficiency.

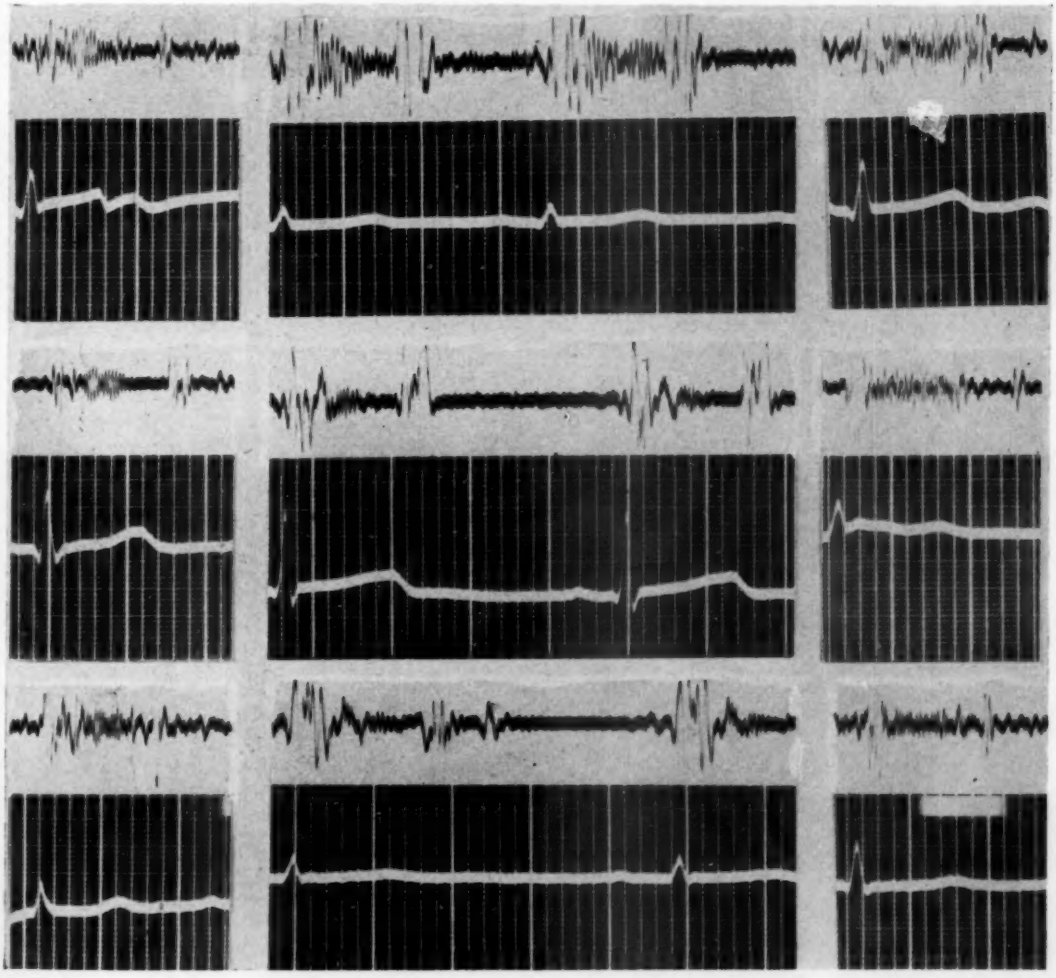

Fig. 1.—Typical phonocardiograms of pulmonic-systolic murmurs in healthy children (shown in the three central tracings). For comparison: three typical phonocardiograms of precordial vibratory ("twanging-string") murmurs from healthy subjects (on left), and three traces from apical systolic murmurs of mitral insufficiency (on right). The peak of the R wave in the electrocardiogram below each phonocardiogram serves to identify the first heart sound.

When traces of pulmonic-systolic murmurs of these respective acoustic characters were compared, no association was observed between the subjective estimate of the degree of harshness of the murmur, on one hand, and the apparent complexity of the wave form, or any other characteristic, on the other.

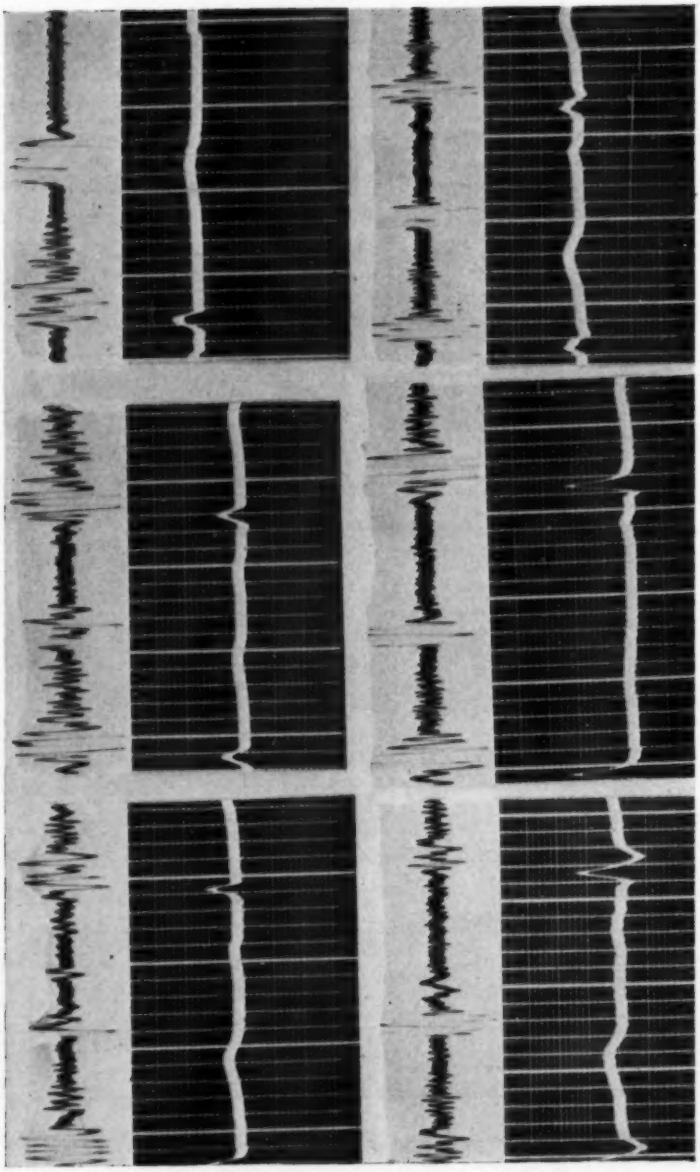

Fig. 2.—Typical phonocardiograms of pulmonic-systolic murmurs graded according to subjective estimates of acoustic quality of harshness on auscultation. Least harsh (on left); moderately harsh (in center); most harsh (on right).

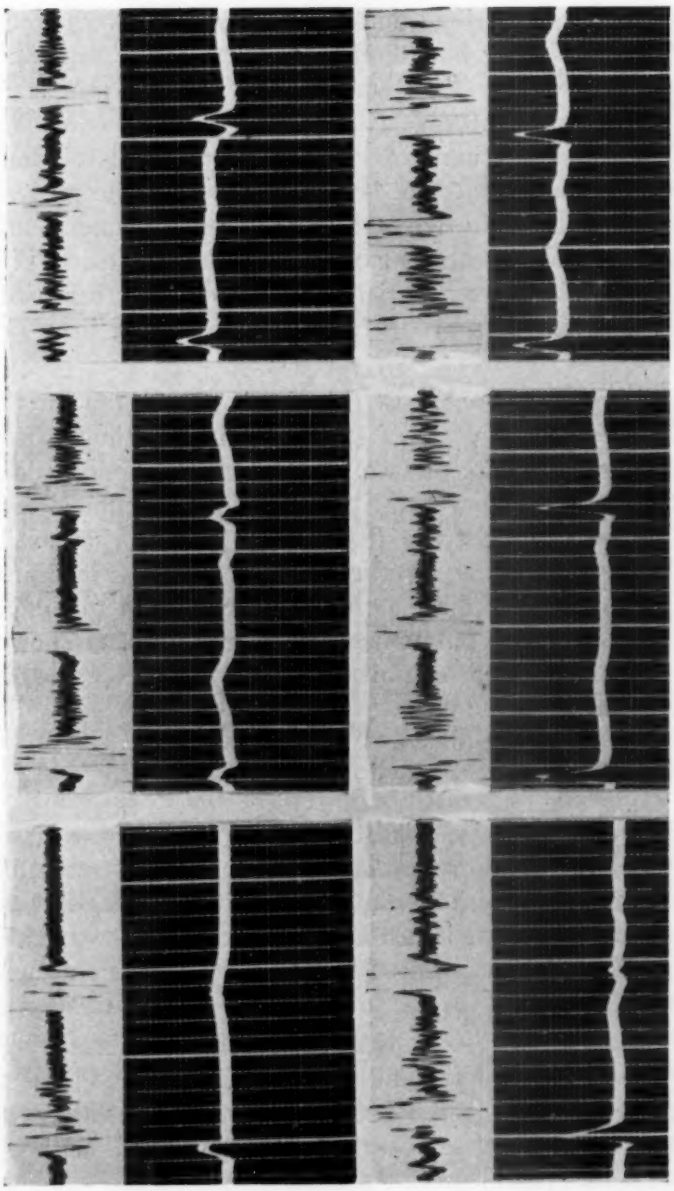

Fig. 3.—Typical phonocardiograms of pulmonic-systolic murmurs in normal children and those with rheumatic fever. On left, from normal children with pulmonic-systolic murmurs; in the center, from children with rheumatic polyarthritis but without clinically detectable carditis; on right, from patients with rheumatic carditis.

Fig. 2 shows examples of traces of pulmonic-systolic murmurs of the least harsh type on the left, moderately harsh in the middle, and most harsh on the right.

4. *Pulmonic-Systolic Murmurs in Rheumatic Fever and Rheumatic Carditis.*—All the murmurs described and reproduced here were from patients in whom extended clinical study had tended to rule out the presence of rheumatic heart disease. However, the pulmonic-systolic murmur can also be heard in many patients with rheumatic fever or rheumatic carditis, although its presence is presumably quite unrelated to the disease, and this murmur was recorded in such situations in order to determine whether the presence of this disease, with or without clinically detectable cardiac involvement, affected the characteristics of this type of murmur. It was found that the traces of pulmonic-systolic murmurs, as obtained from patients with rheumatic infection, did not differ in any observable way as a group from those recorded from presumably normal subjects.

Fig. 3 shows such comparisons. In this figure the traces on the left are from patients with at least five years of study at the Rheumatic Fever Clinic leading to a diagnosis of no heart disease. The central phonocardiograms are from patients with rheumatic polyarthritis but without clinical evidence of carditis. The traces on the right are from patients with rheumatic carditis, each of whom had a typical apical systolic murmur of mitral insufficiency, but one not sufficiently loud to be audible across the precordium to the base of the heart. It will be seen that the pulmonic-systolic murmurs, as shown in these three pairs of phonocardiograms, exhibit the same general characteristics of wave form as were described previously.

DISCUSSION

In the material examined in this study the phonocardiographic tracing produced by the pulmonic-systolic murmur has been found to resemble a simple wave of measurable frequency with sporadic distortions. Since these vibrations were recorded with a moving-string instrument, it is necessary to consider whether the mechanical inertia of the recording device could affect the accuracy of the reproduction of the vibrations.

In the case of the simple vibrations of the precordial vibratory (twanging-string) murmur as shown on the left-hand side of Fig. 1 there is little doubt that this stringed instrument reproduced vibrations essentially as they were transmitted by the microphone, because these vibrations fall well within the range which the instrument can reproduce. As can be seen in the left-hand column of Fig. 1 in comparison with the time scale of the ECG below, the frequency of vibration is of the order of 100 to 120 cycles per second, whereas the instrument employed here can reproduce simple waves of frequencies up to 500 to 600 cycles per second, according to both the manufacturer's specifications and tests carried out in connection with the present studies. (It should also be noted that it would be virtually impossible for an instrument to record a simple wave as an artifact if a complex wave were introduced within the present circumstances

i.e., involving a higher range of frequencies than that of its own natural period, but within a few multiples of the frequency of that simple wave.)

These arguments would also apply in the case of the relatively simple, or uniform, portions of the traces obtained in the case of pulmonic-systolic murmurs, and suggest that to the extent that a simple wave can be discerned in the phonocardiograms of this murmur, the instrument is reproducing vibrations which it is able to follow. It is in the case of the distortions of the simple wave, which are presumably due to superimposed vibrations of other, probably higher, frequency, that we cannot estimate the fidelity with which these are reproduced. In the case of any vibrations of other frequencies it is not possible to estimate by inspection at a point of distortion whether the frequency involved, or the rate of increase of amplitude at that frequency, exceeds the limits of transient response of the recording device. To the extent that these characteristics of the instrument might be exceeded, the amplitudes of the distorting component would fall short of the true value, and the apparent distortion would be correspondingly reduced. The degree of distortion of the discernible basic wave which is seen here is, therefore, a lower limit of the true extent. However, since the maximum frequency which the recording device can reproduce is several multiples of the discernible frequency of the apparently basic wave, it is likely that a substantial portion of the energy of the distorting components is recorded here.

In the study of the precordial "twanging-string" murmur, the interpretation was offered that the simple wave form found in the phonocardiograms suggested as a physical basis for that murmur a vibration of some part of the cardiac tissue at its natural period of vibration, in response to an impulse generated by the hydrodynamic forces of the circulation. It was suggested therefore that "precordial vibratory murmur" might be an appropriate term for that adventitious sound. In the case of the other major nonpathologic murmur, the pulmonic-systolic, the fundamental wave form is sufficiently similar to that of the precordial vibratory murmur to suggest that a major portion of the vibrational energy might have a similar basis. To this fundamental vibration there is, however, evidence of the addition of an undetermined amount of energy of other frequencies which might be either of simple-vibrational types, as in the case of the precordial vibratory murmur, or of a mixed type, as in the case of the blowing murmurs of valvular insufficiency. It is likely that this higher-frequency component or mixture of components contributes less to the total vibrational energy of the murmur than does the basic vibration, since it is known from work on deliberate modulation of sine waves that the admixture of higher-frequency waves or pulses at a lower total level of energy than the basic waves can cause distortion of the latter to the extent that its original pattern can hardly be recognized.

In considering the interpretation just offered, the question should be raised as to whether there is a possibility that the anatomic site at which all these pulmonic-systolic murmurs were recorded might affect the wave form as observed in the traces. Thus, the possibility must not be overlooked that the higher-frequency elements present in the original vibrations might be relatively more

effectively filtered out than the lower-frequency elements by the particular anatomic tissues or structures of the mediastinum before these vibrations reached the microphone, thus leaving the bulk of the energy transmitted by the microphone in the lower-frequency level. Appropriate control material, in this connection, would be provided by tracings of murmurs of known congenital heart disease as recorded at this area. Such material has, in fact, been collected, and such murmurs have been found to yield very complex wave forms. However, since a study by cathode-ray oscillography of several types of cardiac murmurs is currently in progress in this laboratory, it seemed desirable to postpone a comparison of the pulmonic-systolic murmur with the parasternal murmurs of congenital heart disease to the later study, because of the obvious advantages of the cathode-ray oscillograph.

SUMMARY

Phonocardiographic tracings have been taken of the presumably nonpathologic murmurs of children which are heard with greatest intensity in the second intercostal space at the left sternal border (the pulmonic-systolic murmur). Tracings of forty such murmurs have been examined, and the wave forms of all of these have been found to have certain characteristics in common. The form of the pulmonic-systolic murmurs in these tracings was that of an apparently simple wave, of measurable frequency, interrupted by distortions of complex wave form at one or more points in the simple wave. Comparisons are drawn between tracings of these murmurs, those of the precordial vibratory murmur (the "twanging-string" functional murmur of Still), and those of organic murmurs of mitral valvular insufficiency.

SUMMARIO IN INTERLINGUA

Esseva obtenite phonocardiogrammas del presumitemente non-pathologic murmures que se audi in juveniles con un maximo de intensitate in le secunde spatio intercostal al margine sinistro-sternal. Registrationes de 40 tal murmures "pulmonic-systolic" esseva examinate. Il esseva constatate que omne iste registrationes exhibiva certe identic characteristicas del forma de lor undas. In omnes le forma del murmure pulmonic-systolic esseva interrumpite in un o plure punctos per distortiones de forma undic complexe. Es presentate un comparation de iste registrationes con le registrationes de murmures vibratori precordial e con le registrationes del murmure organic de insufficientia del valvula mitral.

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THE ELECTROCARDIOGRAM OF DOGS SURVIVING 1.5° CENTIGRADE

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WHEN the body temperature of dogs is lowered below 20° C., without provision for artificial circulation of oxygenated blood, all animals show progressing degrees of cardiac arrhythmia, and invariably die of ventricular fibrillation.[1] Extracorporeal circulation has been used successfully to prevent death due to ventricular fibrillation. One dog survived after the body temperature had been lowered to 12° C.[2] Electrocardiograms obtained during this study were published.[3]

In other investigations reported from this laboratory it has proved possible to lower the body temperature of adult dogs to 0° C. with long-term survival by using a pump-oxygenator for the refrigeration of blood.[4-6] The unbeating heart thus obtained lends itself most advantageously to open left-heart surgery.[7] The present report deals with the electrocardiographic findings under such conditions.

METHODS AND PROCEDURES

Eight Nembutalized mongrel dogs were cooled to about 28° C. by covering them with crushed ice. Under artificial closed positive pressure respiration system, a left thoracotomy was performed. Forty milligrams per kilogram of quinidine sulfate and 2 milligrams per kilogram of heparin were administered intravenously. Plastic catheters were inserted in the superior and inferior vena cava through the femoral veins for the withdrawal of venous blood. A short plastic catheter was inserted into one femoral artery, and a long catheter was threaded via the other femoral artery into the brachiocephalic artery for the return of the oxygenated blood from the pump-oxygenator.[8] The catheters were connected with the intake and output openings of the instrument and extracorporeal circulation started. The circulating blood could be rapidly cooled or rewarmed by immersing a metal coil in the circuit in cold or warm water. Standard limb electrocardiographic Leads I, II, and III, and aV_R were recorded simultaneously by means of a Sanborn four-channel recorder. Paper speed was 25 millimeters per second throughout. Standardization was checked frequently. Because of the left thoracotomy, precordial leads could not be

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recorded. Lead aV_R was chosen because it proved to be the most stable lead under these experimental conditions. Recordings were taken at each degree centigrade change during the forty minutes of cooling to 5° C., and a continuous recording was made during the fifteen minutes of rewarming to 28° C.

RESULTS

The most striking change observed was the well-known slowing of the heart rate as cooling proceeded (Fig. 1).

Arrhythmia.—The arrhythmias usually observed during hypothermia such as extrasystoles, varying degrees of A-V block, ventricular tachycardia, and auricular and ventricular fibrillation were not seen in these animals as the heart was cooled into asystole.

By an oversight, two dogs did not receive quinidine sulfate until cardiac arrest had occurred, and both animals developed ventricular fibrillation. No attempts were made to defibrillate electrically since cardiac arrest at a lower temperature stopped all heart activity. The electrocardiographic changes during rewarming in these two animals did not differ from those observed in the others.

In one dog auricular fibrillation began at 28° C. and spontaneously changed to nodal rhythm at 18° C. Occasionally, after nodal rhythm has been established there was asystole for one or two beats. During asystole no auricular contractions were observed. Following the next ventricular contraction an auricular contraction was seen and recorded, indicating a true nodal rhythm. After cardiac arrest, sluggish ventricular contractions could be elicited by mechanical stimulation (Fig. 2).

On rewarming, extrasystoles were slightly more frequent than on cooling. The first beats in all but two dogs were followed by a period of asystole which lasted for two to five minutes; then the rhythm became nodal with occasional ventricular extrasystoles without auricular complexes and with brief periods of asystole as noted during cooling. Between 18° and 22° C. the heart went regularly into asystole for about 15 to 120 seconds and then revived in sinus rhythm which persisted for the rest of the rewarming and postoperative period.

Heart Rate.—The heart rate dropped slowly during peripheral cooling and precipitously when blood cooling started (Fig. 3). Cardiac arrest occurred between 12° and 10° C. and lasted from 21 to 35 minutes. When the blood was rewarmed the first heart beats were recorded between 8° to 12° C. The heart rate changed faster than on cooling as temperature rose to 20° C. and, from 20° to about 28° C. more slowly than on cooling.

Electrocardiographic Intervals.—On cooling, the P-R interval (Fig. 4) increased progressively until nodal rhythm supravened. On rewarming, P-R intervals were slightly longer for the same temperature than on cooling.

The QRS complexes showed various forms such as deep S waves, R-R complexes, and splintering. These occurred unpredictably, and frequently two or more patterns were seen in the same dog. No patterns could be distinguished

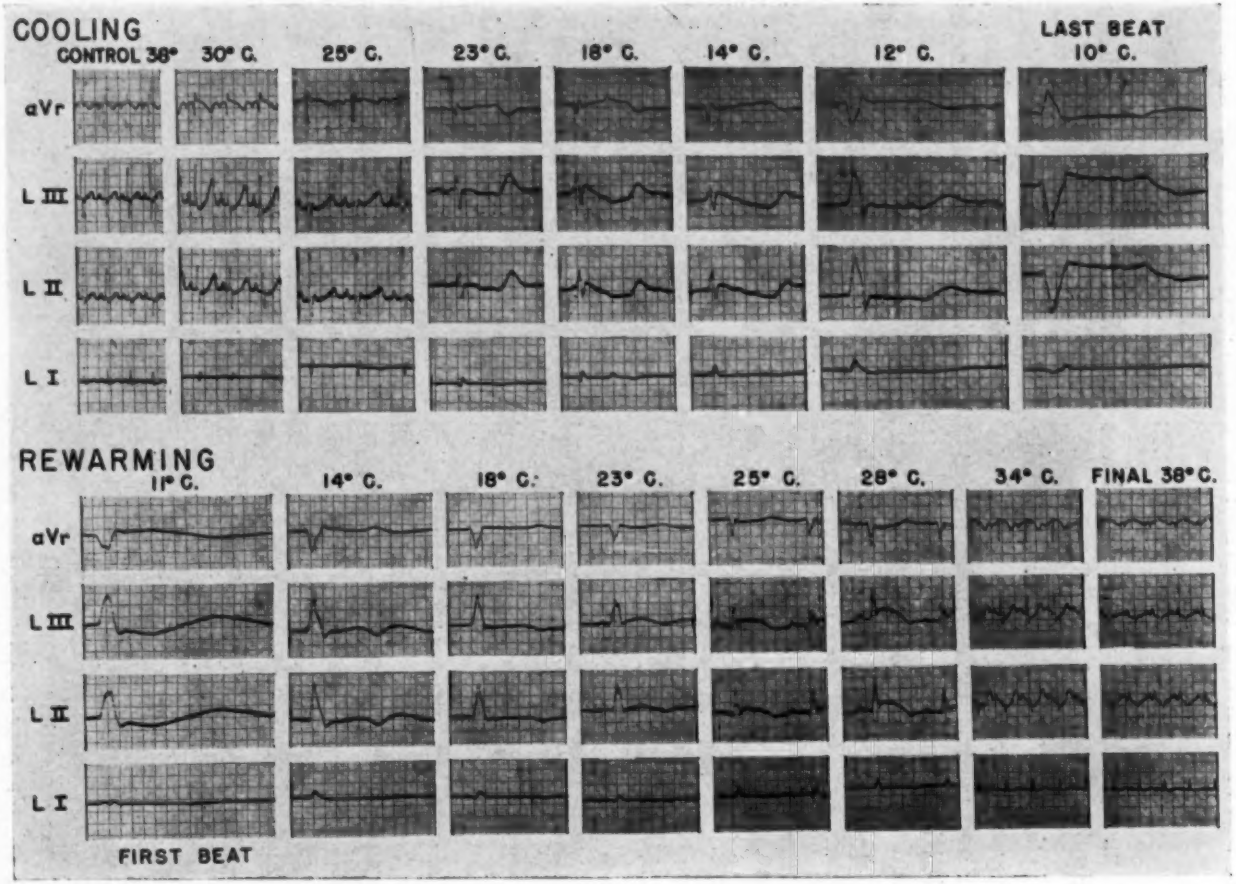

Fig. 1.—Representative electrocardiogram during cooling and rewarming.

HEART RATE

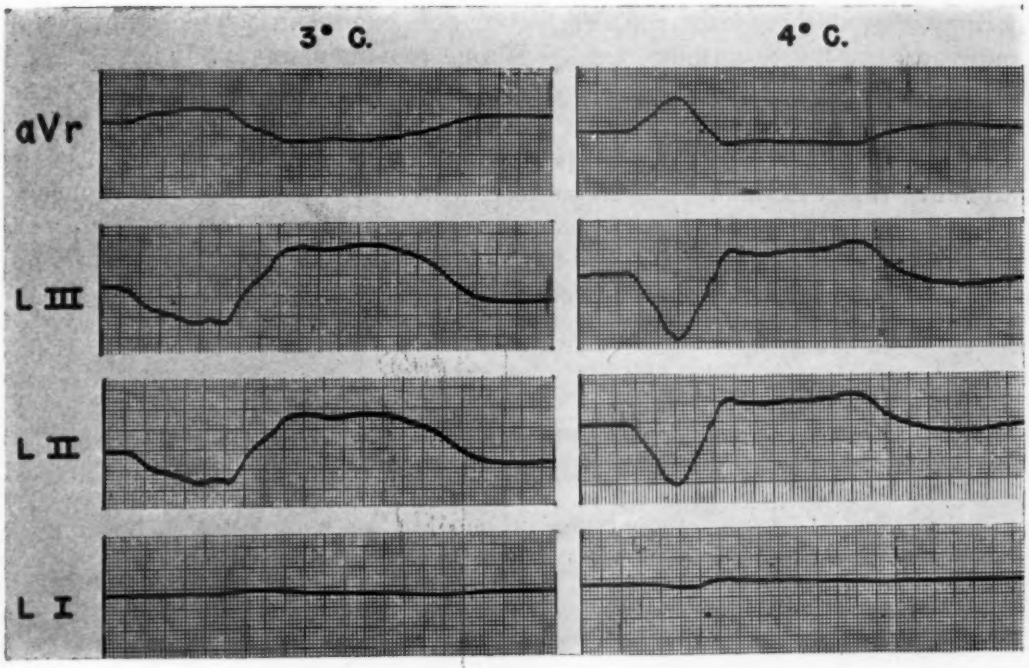

Fig. 2.—Stimulated beats below 10° C.

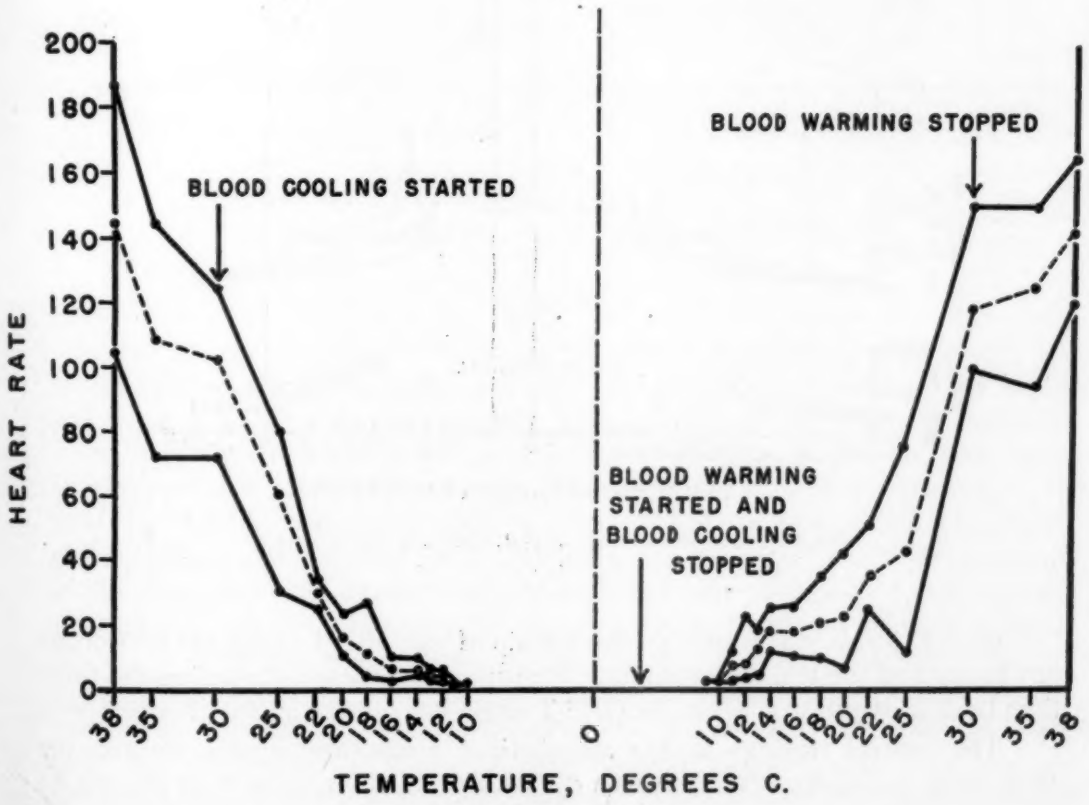

Fig. 3.—Range and average heart rate.

by comparing all three leads and aV_R, as occurs in bundle branch block at normal temperatures. On rewarming, the QRS time remained somewhat longer than during the cooling at comparable temperatures. The Q-T interval underwent by far the greatest change. During cooling to cardiac arrest it increased from about 0.23 sec. to 1.7 sec. From 14° to 10° C. the interval shows such great variability that the small fluctuations in the average curve are probably meaningless. The same holds true for the rewarming period up to about 14° C. In contradistinction to the behaviour of the P-R and QRS intervals the Q-T interval was shorter on rewarming than on cooling.

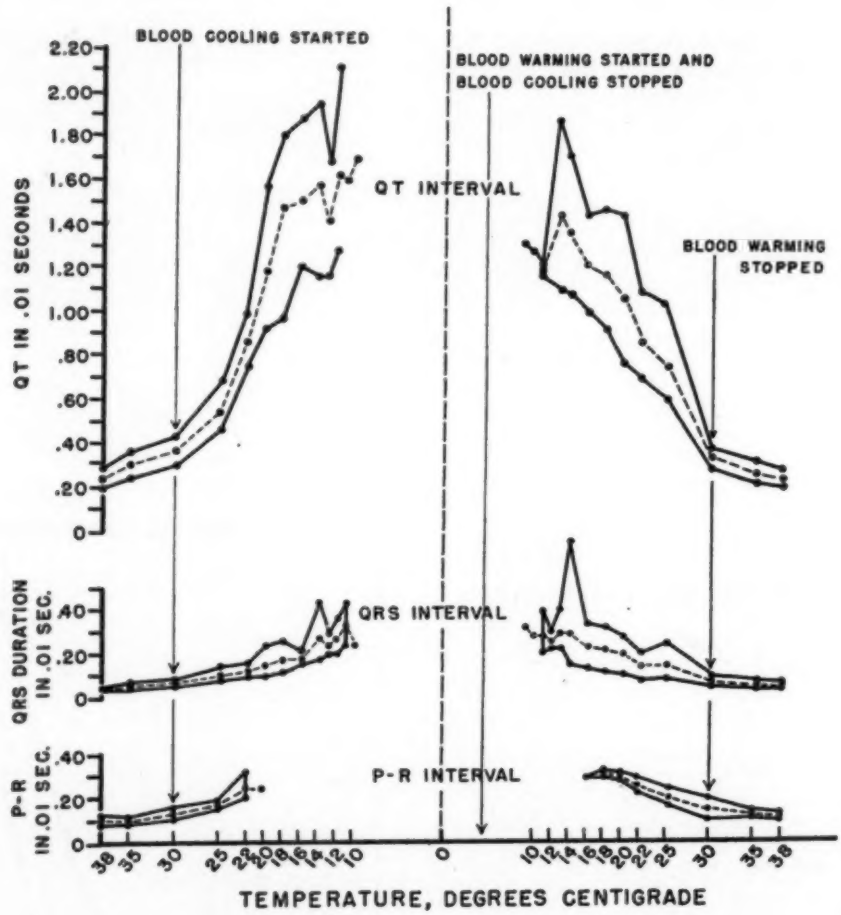

Fig. 4.—Range and average of P-R, QRS and Q-T intervals.

QRS-T Angle.—The spatial relationship of the QRS-T could not be worked out because of the left thoracotomy. However, as a means of relating the three limb leads in the frontal plane the QRS-T angle was plotted against temperature.

The control tracings in the anesthetized normothermic dog showed the same great variability that was seen during cooling to asystole (Fig. 5), and no interpretation can be made. On rewarming, however, the QRS-T angle in

all animals held at about 180° up to a temperature of 25° C. Then, to normal temperature, marked variability was observed.

The initial rewarming period up to 25° C. probably showed an abnormality of repolarization of unknown origin.

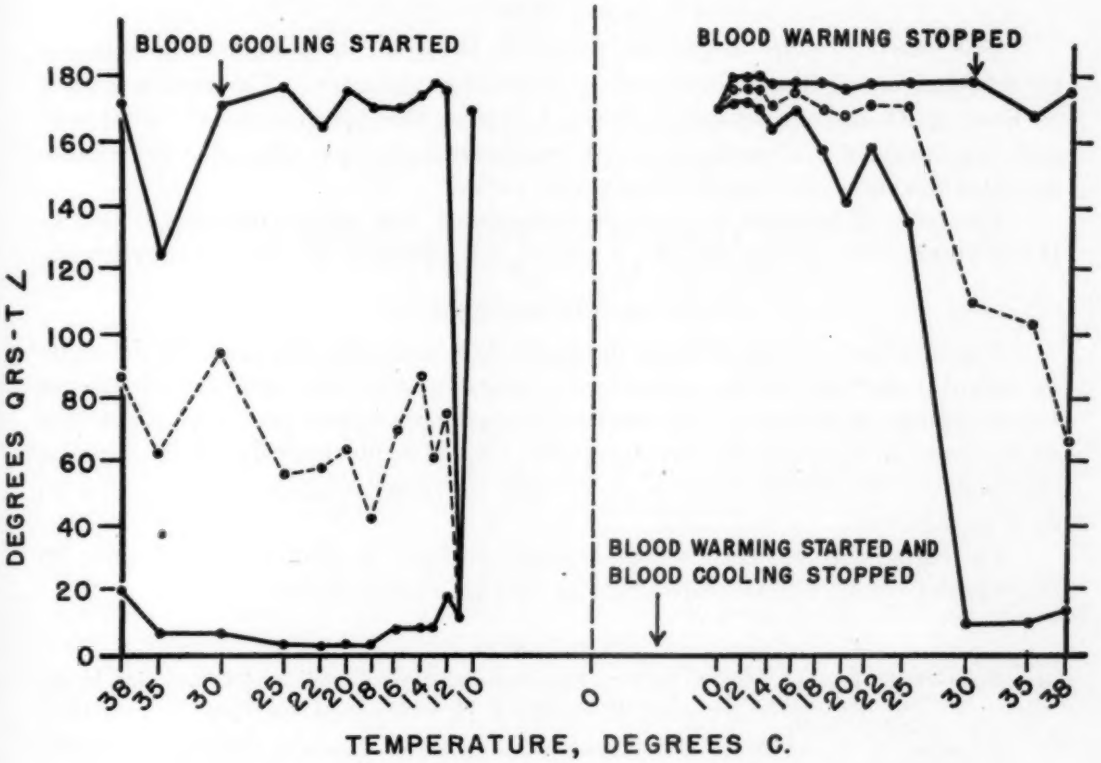

Fig. 5.—Range and average of frontal plane QRS-T angle.

DISCUSSION

The deleterious effect of cold temperature on the activity of the heart has been observed for a long time,[9] and arrhythmias and ventricular fibrillation as the cause of death from hypothermia have received added attention in recent years.[10-12] Survival after lowering of body temperature below the level which is usually lethal for dogs depends upon avoiding some of the known causes of cardiac crisis in prolonged exposure to cold.[6] At no time during the experiment did the heart suffer from anoxia or carbon dioxide retention. The workload of the heart was first partially and then completely taken over by the pump-oxygenator. At the same time the myocardium was perfused with oxygenated blood. It should be noted, however, that the return of oxygenated blood from the instrument was managed in such a way that coronary flow was drastically reduced to the minimal oxygen requirement of a profoundly hypothermic heart in arrest. No increase in venous or right auricular pressure could occur because the entire venous return was shunted into the pump-oxygenator. The increased blood viscosity in hypothermia[12] was prevented by diluting the blood with

Ringer's solution. The administration of quinidine sulfate[13,14] contributed to the decreased irritability of the myocardium as demonstrated by the periods of ventricular fibrillation in the two experiments where quinidine was omitted until hypothermic asystole stopped all heart activity.

SUMMARY

The hearts of eight dogs were cooled to asystole for as long as 35 minutes by refrigeration of blood circulated by a pump-oxygenator. The hearts started to beat again upon rewarming of the blood in the extracorporeal circulation and the animals survived the experiment without any clinically detectable sequelae during a one-month observation period.

Electrocardiographic recordings throughout the entire procedure and in the postoperative period showed a remarkable absence of serious arrhythmias.

SUMMARIO IN INTERLINGUA

Esseva effectuate asystolismo del corde de 8 canes durante periodos de usque 35 minutas per medio de refrigeration extracorporee del sanguine circulante via un pumpa oxygenator. Le cordes recomenciava batter post le recalefaction del sanguine in le circulation extracorporee, e le animales superviveva sin sequelas clinicamente detegibile durante un periodo de observationes postexperimental de 4 septimanas.

Le registrationes electrocardiographic durante e post le experimento se distingueva per un remarcabile absentia de arrhythmias seriose.

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A STUDY OF THE ELECTROCARDIOGRAM AND VECTORCARDIOGRAM IN CONGENITAL HEART DISEASE

III. Electrocardiographic and Vectorcardiographic Findings in Various Malformations

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INTRODUCTION

CONGENITAL cardiac malformations frequently produce modifications of the normal balance of electrical forces generated by the heart, and the recognition and proper interpretation of these may be of considerable differential diagnostic value. Ventricular hypertrophy, one of the most common anatomic findings in congenital heart disease, is primarily responsible for these aberrations in electrical activity.

In previous communications[1,2] we presented an analysis of the electrocardiograms and vectorcardiograms in a group of patients in whom the diagnosis of congenital heart disease with unilateral ventricular hypertrophy was well established. On the basis of this study electrocardiographic and vectorcardiographic criteria for the diagnosis of unilateral ventricular hypertrophy were presented and the relative merits of these criteria assessed. It was concluded that the vectorcardiogram was superior to the electrocardiogram in detecting the type of unilateral ventricular hypertrophy present.

It is the purpose of the present report to analyze and to evaluate the electrocardiogram and vectorcardiogram in each specific malformation studied.

MATERIAL AND METHODS

One hundred and thirty-five patients with congenital heart disease in whom the diagnosis appears to be well established form the basis of this study. Table I indicates the diagnoses and age distributions of these patients. Detailed

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descriptions of the manner in which the diagnoses were made and of the methods of recording the electrocardiograms and vectorcardiograms were presented previously.[1,2] Briefly, however, the electrocardiograms were all recorded on a three-channel direct-writing cardiograph at various paper speeds and standardizations to facilitate interpretation. The vectorcardiograms were obtained using the cube method of electrode placement.[3]

TABLE I. DISTRIBUTION OF CASES BY DIAGNOSIS AND AGE

DIAGNOSIS	AGE									
	UNDER 1 YR.	1-2 YR.	3-5 YR.	6-8 YR.	9-11 YR.	12-14 YR.	15-19 YR.	20-30 YR.	OVER 30 YR.	TOTAL
Tetralogy of Fallot	—	—	10	5	5	3	1	1	1	26
Pulmonic stenosis	—	—	1	6	3	3	4	1	2	20
Interatrial septal defect	—	—	3	2	3	1	3	3	4	19
Tricuspid atresia	—	—	3	1	—	1	—	—	—	5
Coarctation of the aorta	—	—	—	1	2	1	5	—	2	11
Congenital aortic or subaortic stenosis	—	—	5	3	—	1	3	—	—	12
Interventricular septal defect	1	1	2	3	4	1	—	—	—	12
Eisenmenger's complex	—	—	3	—	2	2	1	1	—	9
Patent ductus arteriosus	1	1	3	—	1	1	2	2	1	12
Idiopathic dilatation of pulmonary artery	—	—	—	—	—	2	1	1	—	4
Aortic septal defect	—	—	—	—	—	—	1	—	—	1
Aberrant pulmonic veins	1	—	—	—	—	—	—	—	—	1
Dextrocardia	—	—	—	1	—	—	—	1	—	2
Levocardia	—	—	1	—	—	—	—	—	—	1
Totals	3	2	31	22	20	16	21	10	10	135

The electrocardiographic criteria employed here in the diagnosis of ventricular hypertrophy are those defined in the first paper of this series[1] and are based on the limits of normal voltage and ventricular activation time compiled by Kossmann.[4] The diagnosis of ventricular hypertrophy may be reliably established when one or more of these normal limits are exceeded and if the electrocardiogram presents no evidence of conduction disturbances, of an RSR′ pattern over right precordial leads, or of myocardial infarction. The numerical values of these limits are presented in Table II. Since we have studied only a limited number of patients below one year of age, these criteria require further evaluation in this group. The electrocardiographic criteria employed in the diagnosis of right bundle branch block are those of Wilson and associates.[5]

TABLE II. LIMITS OF NORMAL VOLTAGE IN THE ELECTROCARDIOGRAM*

AGE (YR.)	R_{V1} MAXIMUM NORMAL VOLTAGE IN MM.	R_{V1} MINIMUM NORMAL VOLTAGE IN MM.	S_{V1} MAXIMUM NORMAL VOLTAGE IN MM.	S_{V1} MINIMUM NORMAL VOLTAGE IN MM.	R_{V6} MAXIMUM NORMAL VOLTAGE IN MM.	R_{V6} MINIMUM NORMAL VOLTAGE IN MM.
Under 1	29.0	3.0	28.0	0.0	24.0	0.0
1–10	20.0	0.4	36.5	0.0	28.0	5.0
10–20	16.7	0.4	26.6	0.0	25.0	3.5
Over 20	15.5	0.0	26.2	0.8	22.6	2.0

AGE (YR.)	S_{V6} MAXIMUM NORMAL VOLTAGE IN MM.	R_{V3R} MAXIMUM NORMAL VOLTAGE IN MM.	R_{aVR} MAXIMUM NORMAL VOLTAGE IN MM.	S_{aVR} MAXIMUM NORMAL VOLTAGE IN MM.	R_{aVL} MAXIMUM NORMAL VOLTAGE IN MM.	
Under 1	30.0		9	18.0	10.0	
1–10	13.0	9.9	19.5	19.5	11.8	
10–20	11.3	8.0	8.0	17.0	10.1	
Over 20	14.3		3.0	11.0	10.1	

The diagnosis of right ventricular hypertrophy is made when an electrocardiogram presents an R_{V1}, S_{V6}, R_{V3R}, or R_{aVR} taller than the maximum normal amplitude, an S_{V1}, or R_{V6} smaller than the minimum normal amplitude, or an intrinsicoid deflection in V_1 occurring 0.04 sec. or later.

The diagnosis of left ventricular hypertrophy is made when an electrocardiogram presents an S_{V1}, R_{V6}, S_{aVR}, or R_{aVL} taller than the maximum normal amplitude, an R_{V1} smaller than the minimum normal amplitude, or an intrinsicoid deflection in V_6 occurring 0.06 sec. or later.

***Compiled by Kossmann.[4]**

We have found the vector loop in the horizontal plane to be the most important in the diagnosis of ventricular hypertrophy. In right ventricular hypertrophy the loop is inscribed in a clockwise fashion,[2,6] whereas in the normal,[7] and in left ventricular hypertrophy[8] the direction of inscription is counterclockwise. In right ventricular hypertrophy the vector loop is usually oriented to the right, while in left ventricular hypertrophy it is directed to the left and posteriorly.

RESULTS

The maximum amplitudes of normal P waves in Leads II and V_1 are shown in Table III, which is obtained from Kossmann's compilation of several series of normal electrocardiograms.[4] The maximum normal duration of the P wave is considered to be 0.10 sec. Table IV indicates the frequency of the various P-wave abnormalities in this series.

TABLE III. MAXIMUM NORMAL AMPLITUDE OF P WAVES IN MM.*

AGE (YR.)	LEAD II	LEAD V_1
Under 1	2.5	2.5
1–10	3.0	2.5
10–20	2.1	2.2
Over 20	3.0	2.2

*Compiled by Kossmann.[4]

The electrocardiographic and vectorcardiographic diagnoses for each malformation studied are listed in Table V.

Table VI represents the deviations of the electrical axes of the QRS derived from the standard leads.

DISCUSSION

Tetralogy of Fallot.—Abnormal P waves have been frequently observed in the electrocardiograms of patients with the tetralogy of Fallot. Tall, peaked P waves have been found in 20 to 96 per cent of the patients in various series,[9-12] and presumably indicate the presence of right atrial hypertrophy. Approximately one-third of our patients presented P waves exceeding the limits of normal (Table IV). These abnormalities consisted chiefly of tall P waves in Lead V_1.

Many observers have commented on the frequency of right-axis deviation in the tetralogy of Fallot. Donzelot and associates[10] noted this in 198 of 200 patients. Soulié and colleagues[13] and Brown[14] have stated that the finding of left, or of no axis deviation should exclude the diagnosis of the tetralogy of Fallot. On a theoretical basis, however, one would not expect to find only right-axis deviation in patients with this malformation. We have found that in right ventricular hypertrophy the horizontal plane contains the diagnostic vectorcardiographic pattern, and identical vector loops in this plane may be associated with widely varying frontal plane vector orientations.[2] Since the mean electrical axis of the QRS, determined from the standard electrocardiographic leads, is

TABLE IV. P-WAVE ABNORMALITIES IN VARIOUS CONGENITAL CARDIAC MALFORMATIONS

DIAGNOSIS	NO. OF PATIENTS	LEAD II				LEAD V_1			
		ABNORMALLY TALL	ABNORMALLY BROAD	PEAKED	NOTCHED	ABNORMALLY TALL	ABNORMALLY BROAD	PEAKED	NOTCHED
Tetralogy of Fallot	26	5	2	4	1	7	0	8	0
Pulmonic stenosis	20	2	1	3	1	2	0	4	0
Interatrial septal defect	19	1	8	1	0	1	0	0	0
Tricuspid atresia	5	2	2	3	0	1	0	2	0
Coarctation of the aorta	11	0	3	1	0	0	0	0	0
Subaortic or aortic stenosis	12	0	2	2	0	0	1	1	0
Interventricular septal defect	12	2	4	2	0	0	0	1	0
Eisenmenger's complex	9	1	4	1	1	1	2	0	0
Patent ductus arteriosus	12	1	1	1	1	0	1	2	0
Idiopathic dilatation of pulmonary artery	4	0	1	0	0	0	0	0	0
Aortic septal defect	1	0	0	0	0	0	0	0	0
Aberrant pulmonic veins	1	0	0	1	0	0	0	1	0
Dextrocardia	2	0	0	0	0	0	0	0	0
Levocardia	1	0	1	0	0	0	1	0	0

TABLE V. ELECTROCARDIOGRAPHIC AND VECTORCARDIOGRAPHIC DIAGNOSES IN VARIOUS CONGENITAL CARDIAC MALFORMATIONS

DIAGNOSIS	NO. OF PATIENTS	ELECTROCARDIOGRAM						VECTORCARDIOGRAM		
		NORMAL	RIGHT VENTRICULAR HYPERTROPHY	LEFT VENTRICULAR HYPERTROPHY	COMBINED VENTRICULAR HYPERTROPHY	RIGHT BUNDLE BRANCH BLOCK†	RSR'‡	NORMAL	RIGHT VENTRICULAR HYPERTROPHY	LEFT VENTRICULAR HYPERTROPHY
Tetralogy of Fallot	26	2	21	0	0	1	2	1	25	0
Pulmonic stenosis	20	6	10	0	0	2	2	0	20	0
Interatrial septal defect	19	2	9	0	0	7	1	0*	18	0
Tricuspid atresia	5	1	0	4	0	0	0	1	0	4
Coarctation of the aorta	11	7	0	4	0	0	0	4	0	7
Subaortic or aortic stenosis	12	10	0	2	0	0	0	7	0	5
Interventricular septal defect	12	5	2	1	2	2	0	10*	0	1
Eisenmenger's complex	9	0	7	10	0	1	0	0	7	2
Patent ductus arteriosus	12	6	5	1	0	0	0	8	2	2
Idiopathic dilatation of the pulmonary artery	4	3	0	0	0	1	0	4	0	0
Aortic septal defect	1	0	0	1	0	0	0	0	0	1
Aberrant pulmonary veins	1	0	1	0	0	0	0	0	1	0
Dextrocardia	2	1	1	0	0	0	0	2	0	0
Levocardia	1	0	1	0	0	0	0	0	1	0

*One patient with an interatrial septal defect, and one with an interventricular septal defect had vectorcardiograms showing right bundle branch block.
†Incomplete or complete right bundle branch block.
‡RSR′ in right precordial leads with QRS duration less than 0.08 sec.

related to the orientation of the vector loop in the frontal plane only, it follows that in the electrocardiograms of patients with right ventricular hypertrophy the mean electrical axes might be expected to show wide variation. In the twenty-six patients with the tetralogy of Fallot, two had no axis deviation while four had left-axis deviation (Table VI).

Right ventricular hypertrophy was correctly diagnosed by the electrocardiogram in twenty-one instances, and by the vectorcardiogram in all but one case. It is of interest that twenty-four out of the twenty-six of the vector loops in these patients with the tetralogy of Fallot belong to the Group 2 pattern, as previously defined.[2] It is possible that this observation may prove of differential diagnostic significance.

TABLE VI. MEAN ELECTRICAL AXIS OF QRS IN VARIOUS CONGENITAL CARDIAC MALFORMATIONS

DIAGNOSIS	0° TO 90° NO AXIS DEVIATION	90° TO 180° RIGHT-AXIS DEVIATION	0° TO −180° LEFT-AXIS DEVIATION	TOTAL
Tetralogy of Fallot	2	20	4	26
Pulmonic stenosis	5	11	4	20
Interatrial septal defect	4	8	7	19
Tricuspid atresia	2	—	3	5
Coarctation of the aorta	7	2	2	11
Subaortic or aortic stenosis	11	—	1	12
Interventricular septal defect	9	1	2	12
Eisenmenger's complex	2	6	1	9
Patent ductus arteriosus	9	3	—	12
Idiopathic dilatation of pulmonary artery	4	—	—	4
Aortic septal defect	1	—	—	1
Aberrant pulmonic veins	1	—	—	1
Dextrocardia	1	—	1	2
Levocardia	—	1	—	1

Pulmonic Stenosis With Normal Aortic Root.—Only three of the twenty patients with pulmonic stenosis had abnormal P waves. Each of these had markedly elevated right ventricular systolic pressures, greater than 120 mm. Hg. While right-axis deviation has been stressed as a common finding in pulmonic stenosis[14,15] the presence of left or of no axis deviation has also been reported.[16-18] As in the tetralogy of Fallot, we believe that the absence of right-axis deviation does not exclude the diagnosis of pulmonic stenosis. Of our twenty patients, only eleven had right-axis deviation, five presented no axis deviation, while four had left-axis deviation. The presence of right ventricular hypertrophy was again reflected more accurately by the vectorcardiogram (19 of 20 patients) than by the electrocardiogram (10 of 20 patients).

Some investigators[11,17] have noted a correlation between electrocardiographic and hemodynamic findings, those patients with the highest right ventricular systolic pressures exhibiting the more advanced electrocardiographic changes. In this series, however, there was no consistent correlation between the level of the right ventricular systolic pressure and the electrocardiographic or vector-

cardiographic findings, except for the P-wave abnormalities noted previously. While the average of the right ventricular systolic pressures in the patients with concordant T loops in the vectorcardiogram was 73 mm. Hg, and 110 mm. Hg in the patients with discordant T loops, there was considerable overlapping of pressures in the individual cases.

Interatrial Septal Defect.—The presence of P-wave abnormalities in interatrial septal defect has been noted by several observers.[14,19,20] Eight of the nineteen patients in this series had abnormally wide P waves, i.e., greater than 0.10 sec. duration. Seven of the nineteen patients with interatrial septal defect had left-axis deviation, a finding which has been noted by others.

Barber and associates[21] have reported a high incidence (over 90 per cent) of the electrocardiographic patterns of so-called right bundle branch block in this malformation. However, only seven of the nineteen patients revealed right bundle branch block patterns electrocardiographically while an eighth patient had an RSR′ configuration in V_1 with a total QRS duration of 0.07 sec. It is true that the electrocardiographic pattern of right bundle branch block occurred more frequently in interatrial septal defect than in any of the other malformations which were studied, but we cannot agree that the diagnosis of interatrial septal defect is untenable in the absence of this electrocardiographic finding.[21]

When the so-called right bundle branch block pattern occurs in the electrocardiograms of patients with interatrial septal defect (and other congenital malformations producing right ventricular hypertrophy), the vectorcardiogram, according to our criteria,[2] usually does not confirm the existence of a conduction disturbance. In six of the seven patients with interatrial septal defect whose electrocardiograms revealed so-called right bundle branch block, the vectorcardiograms showed only right ventricular hypertrophy. Only one patient had vectorcardiographic evidence of right bundle branch block. The so-called right bundle branch block electrocardiographic pattern in patients with lesions producing right ventricular hypertrophy, as has been indicated previously,[1,2,6] results merely from the particular configuration of the early portion of the QRS vector loop in the horizontal plane. Recent observations have confirmed this interpretation of the vectorcardiograms.[22]

Congenital Subaortic or Aortic Stenosis.—Left-axis deviation in congenital subaortic or aortic stenosis has been reported.[14,15,23] Only one of our twelve patients had left-axis deviation. We have stressed that both the electrocardiogram and the vectorcardiogram are of relatively limited value in detecting the presence of left ventricular hypertrophy in congenital malformations, albeit the latter is the more reliable technique.[2] Thus, only two of the twelve electrocardiograms revealed left ventricular hypertrophy while five vectorcardiograms did so. Wenger and Wick[24] noted left ventricular hypertrophy in the vectorcardiograms of both of their patients with this lesion.

Coarctation of the Aorta.—Gross[25] and Christensen and Hines[26] concluded that the electrocardiogram in coarctation of the aorta is useful in evaluating the degree of myocardial strain or damage. Both Soulié[13] and Metianu and

their associates[27] stated that the presence of marked electrocardiographic changes of left ventricular hypertrophy in patients with coarctation of the aorta may indicate the presence of an associated aortic valvular lesion. The latter observers noted normal electrocardiograms in seven out of forty-one patients with coarctation, while the other thirty-four patients showed left ventricular hypertrophy. Sixteen of the forty-one patients revealed a conduction defect, six with right bundle branch block and ten with left bundle branch block.[27] Ziegler[28] has recently correlated the electrocardiographic findings with the relative positions of the ductus arteriosus and the coarctation, as well as with the presence of patency of the ductus in the presence of coarctation.

In our eleven patients, only four had electrocardiograms indicating left ventricular hypertrophy. The vectorcardiogram again reflected the presence of the existing anatomic hypertrophy somewhat more often (Fig. 1), diagnosing left ventricular hypertrophy in seven instances. There was no correlation between the age of the patient and the electrocardiographic or vectorcardiographic findings.

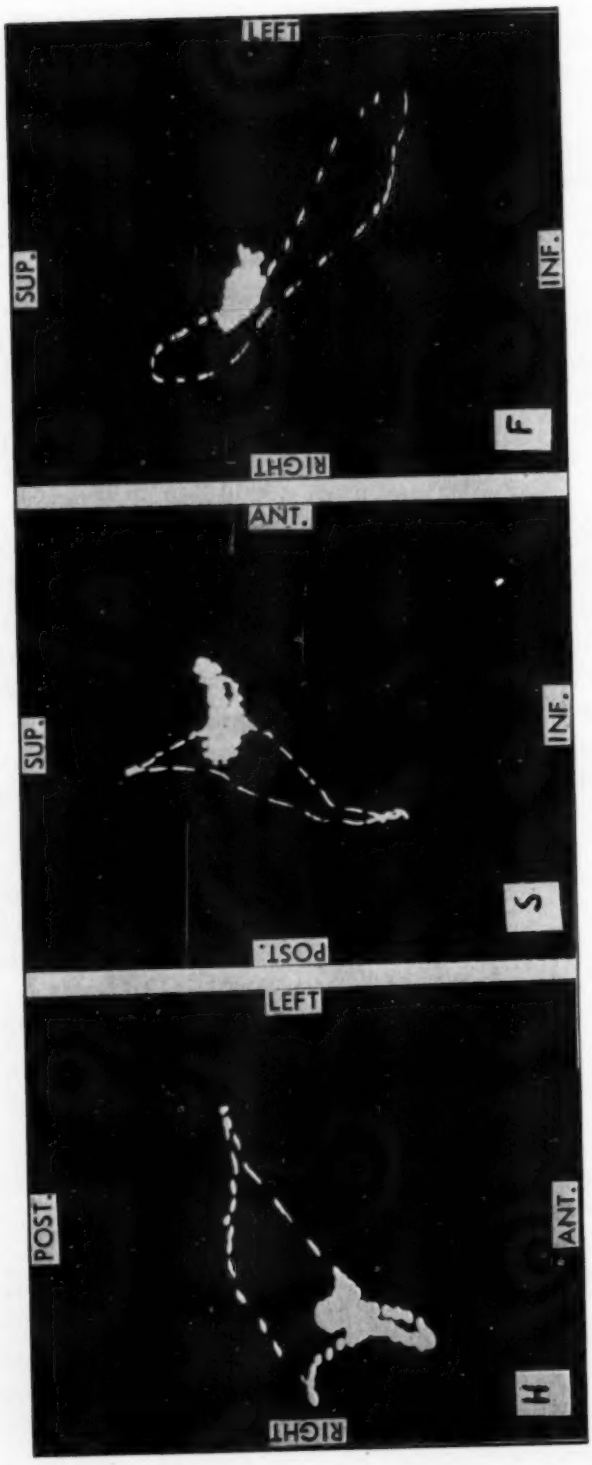
Tricuspid Atresia.—Left-axis deviation has been reported to be a very frequent electrocardiographic finding in tricuspid atresia.[14,15,29-32] Donzelot and associates state that 85 to 90 per cent of these patients present with left-axis deviation. Indeed, tricuspid atresia has been considered the sole malformation in which cyanosis and left-axis deviation coexist. Table VII, however, presents other conditions in which this combination has been found. In this series of 135 patients with congenital heart disease, there were four patients with tetralogy of Fallot and one with Eisenmenger's complex who had left-axis deviation and cyanosis.

TABLE VII. CONGENITAL CARDIAC MALFORMATIONS (OTHER THAN TRICUSPID ATRESIA) IN WHICH CYANOSIS AND LEFT-AXIS DEVIATION HAVE BEEN FOUND TO COEXIST

(1)	Pulmonic stenosis with atrial septal defect[16]
(2)	Partial transposition of the great vessels with pulmonic stenosis[33]
(3)	Truncus arteriosus[14,30]
(4)	Tetralogy of Fallot with interatrial septal defect[34]
(5)	Single ventricle[14]
(6)	Aortic atresia[35]
(7)	Eisenmenger's complex[36]
(8)	Complete transposition of the great vessels[30]
(9)	Ebstein's anomaly[33]
(10)	Infantile type of coarctation of the aorta[33]

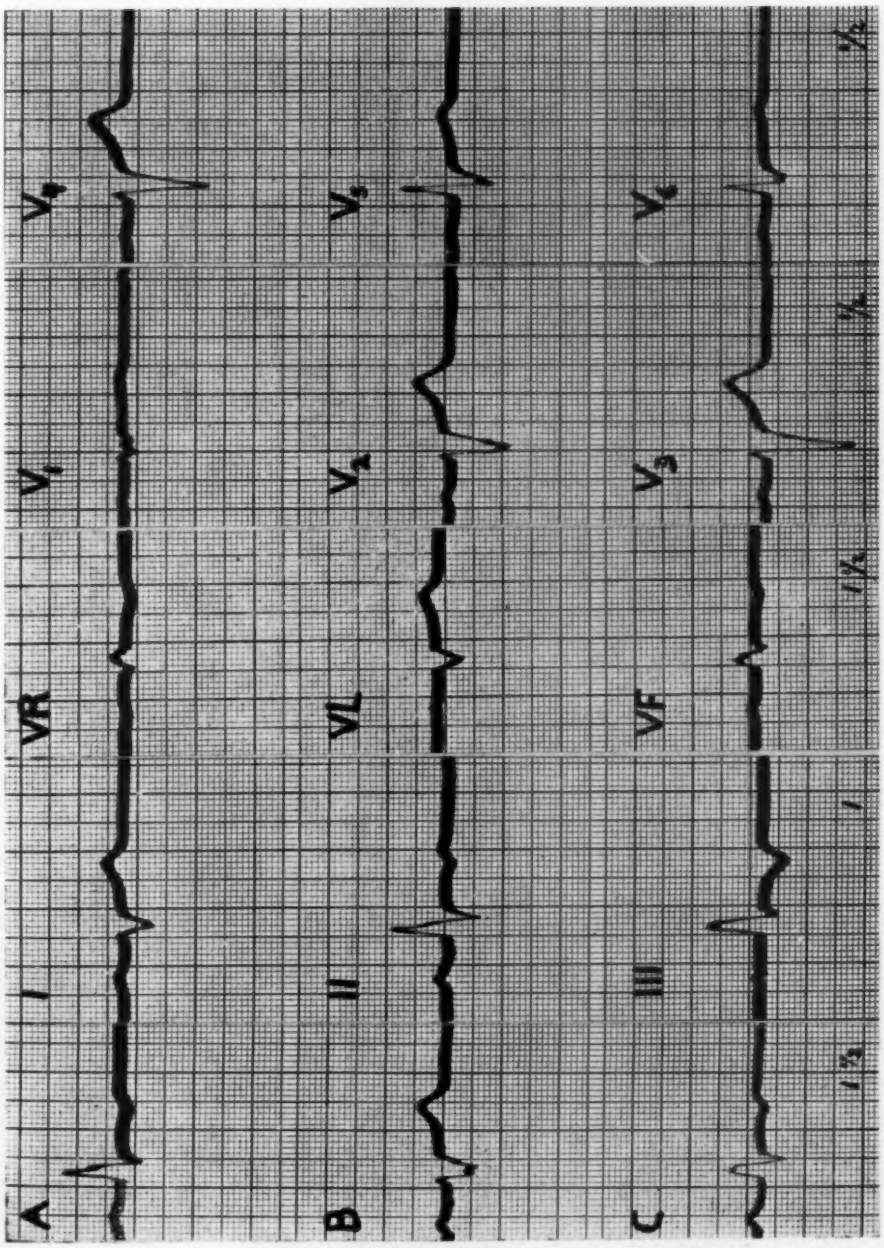
It is of interest that tricuspid atresia with right-axis deviation has also been reported.[36] Of our five patients with this lesion, three presented left-axis deviation and two no axis deviation.

Donzelot and associates[32] stated that all patients with tricuspid atresia have electrocardiographic evidence of left ventricular hypertrophy and 25 per cent have left bundle branch block. Four of our five patients had evidence of



A.

Fig. 1, *A*.—*(For legend see opposite page.)*



B.

Fig. 1.—An 18-year-old male with coarctation of the aorta. The vectorcardiogram (*A*) reveals left ventricular hypertrophy as evidenced by displacement of the QRS vector loop posteriorly and to the left and by discordancy of the T loop. The electrocardiogram (*B*) taken at twice the standard paper speed shows no ventricular hypertrophy with right-axis deviation. Figures at the bottom indicate standardization factors.

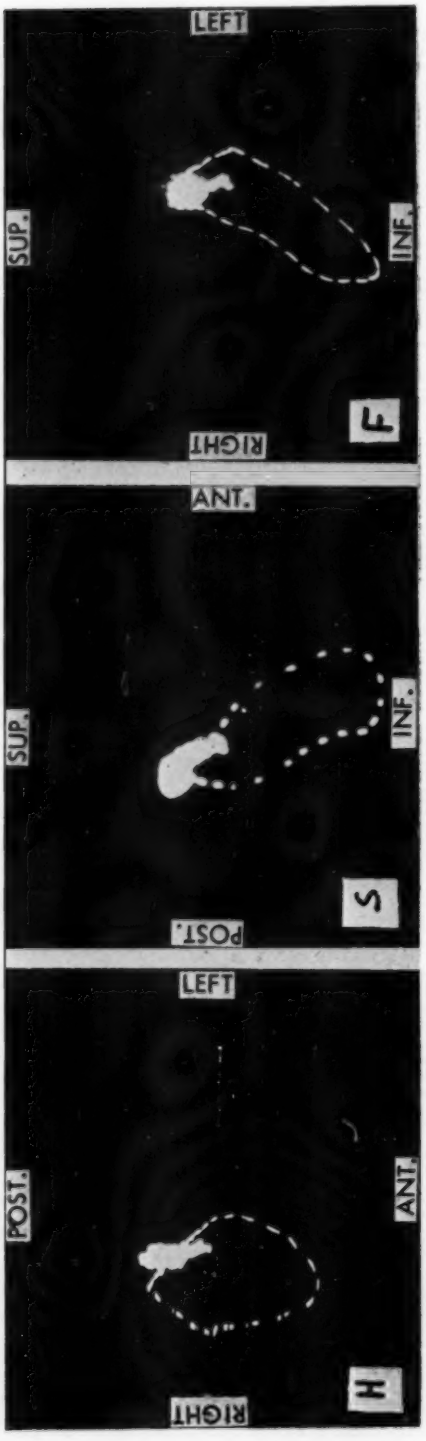


Fig. 2.—A 24-year-old female with patent ductus arteriosus, pulmonary hypertension, and reversal of flow. The vectorcardiogram indicates right ventricular hypertrophy as evidenced by displacement of the QRS loop anteriorly and to the right. There is clockwise inscription in the horizontal plane which is characteristic of right ventricular hypertrophy.

left ventricular hypertrophy in the electrocardiogram and vectorcardiogram, while the remaining one showed no abnormality. Three patients in this group exhibited abnormally tall P waves.

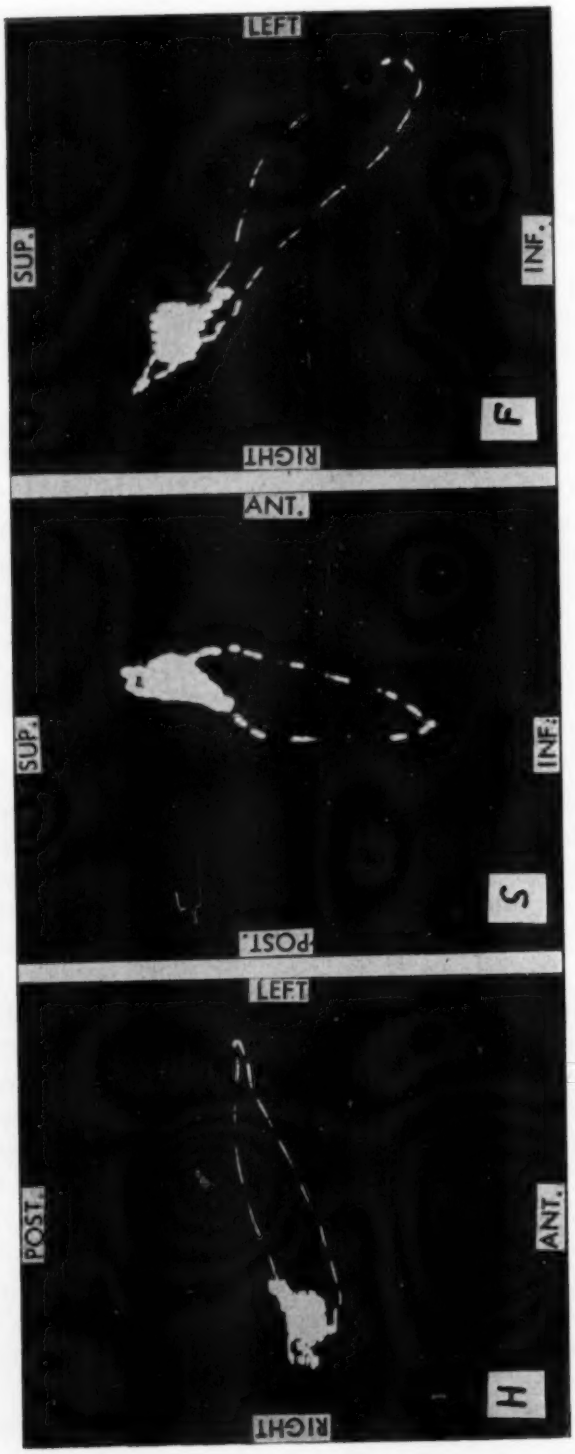
Patent Ductus Arteriosus.—The vast majority of patients with patent ductus arteriosus have normal electrocardiograms and no deviation of the electrical axis. Brown[14] and Gross and Longino[37] believe that the presence of left-axis deviation should arouse suspicion of a large left-to-right ductal shunt. Less than 1 per cent of the patients in Gross and Longino's[37] series presented right-axis deviation, and the authors indicate that in its presence an associated malformation, or pulmonary hypertension, should be considered. In those patients with pulmonary hypertension sufficient to cause reversal of normal ductal flow, the finding of right ventricular hypertrophy in the electrocardiogram is nearly always present.[38-40]

Nine of the twelve patients with patent ductus arteriosus revealed no axis deviation, while three had right-axis deviation. Of the latter group, one patient had marked pulmonary hypertension with reversal of flow and cyanosis (Fig. 2). Another, a 3-month-old infant, presented with embolic occlusion of the pulmonary artery in addition to the large patent ductus arteriosus at post-mortem examination. The third patient was a 4-year-old with an uncomplicated patent ductus arteriosus.

The vectorcardiograms correlated well with the various hemodynamics encountered. Thus, the two patients with right ventricular hypertension showed right ventricular hypertrophy (Fig. 2), while the two patients with the largest left-to-right ductal flow had left ventricular hypertrophy.

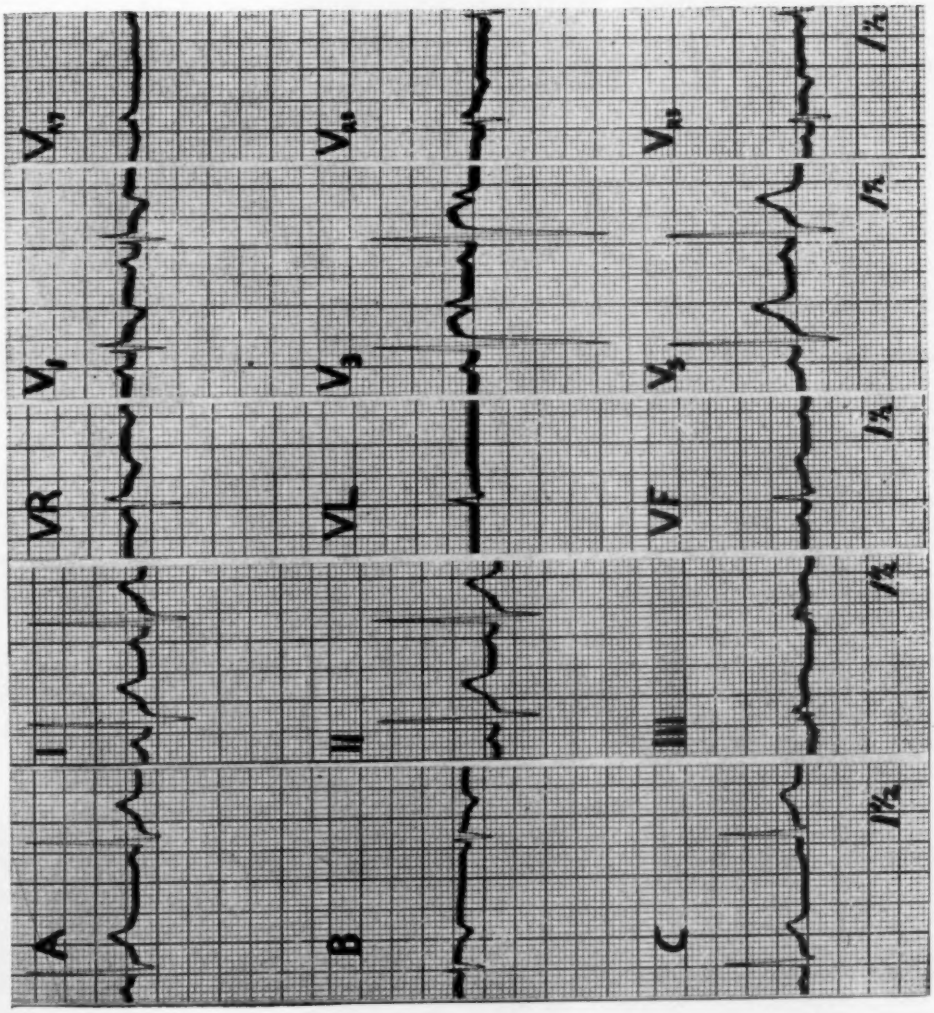
Interventricular Septal Defect.—The patients in this category had no evidence of arterial unsaturation and had normal or only moderately elevated pulmonary artery pressure. In this congenital anomaly, the electrocardiogram is generally reported to be normal.[14,15,41] Nevertheless, only three of the twelve patients with uncomplicated interventricular septal defect had entirely normal electrocardiograms. The other nine patients exhibited a wide variety of patterns, i.e., right ventricular hypertrophy, left ventricular hypertrophy, the combination of right and left ventricular hypertrophy, and incomplete right bundle branch block. The vectorcardiogram, however, showed a normal balance of electrical forces in ten of the twelve patients. In the remaining two patients, the vectorcardiogram confirmed the electrocardiographic diagnosis of right bundle branch block in one, and of left ventricular hypertrophy in the other. No patient in this group presented right ventricular hypertrophy in the vectorcardiogram. Wenger and Wick[24] have also reported normal vectorcardiograms in three patients with interventricular septal defects. There is an increased burden presented to both ventricles in this malformation, and the finding of a normal vectorcardiogram merely indicates that the normal balance of electrical forces is maintained.

Eisenmenger's Complex.—In accordance with Seltzer's functional approach,[42] Eisenmenger's complex is defined as the combination of interventricular septal defect, marked pulmonary hypertension, equal to systemic pressure levels, and diminished arterial oxygen saturation. Anatomically, it may be difficult to decide whether or not there is actual overriding of the aorta.



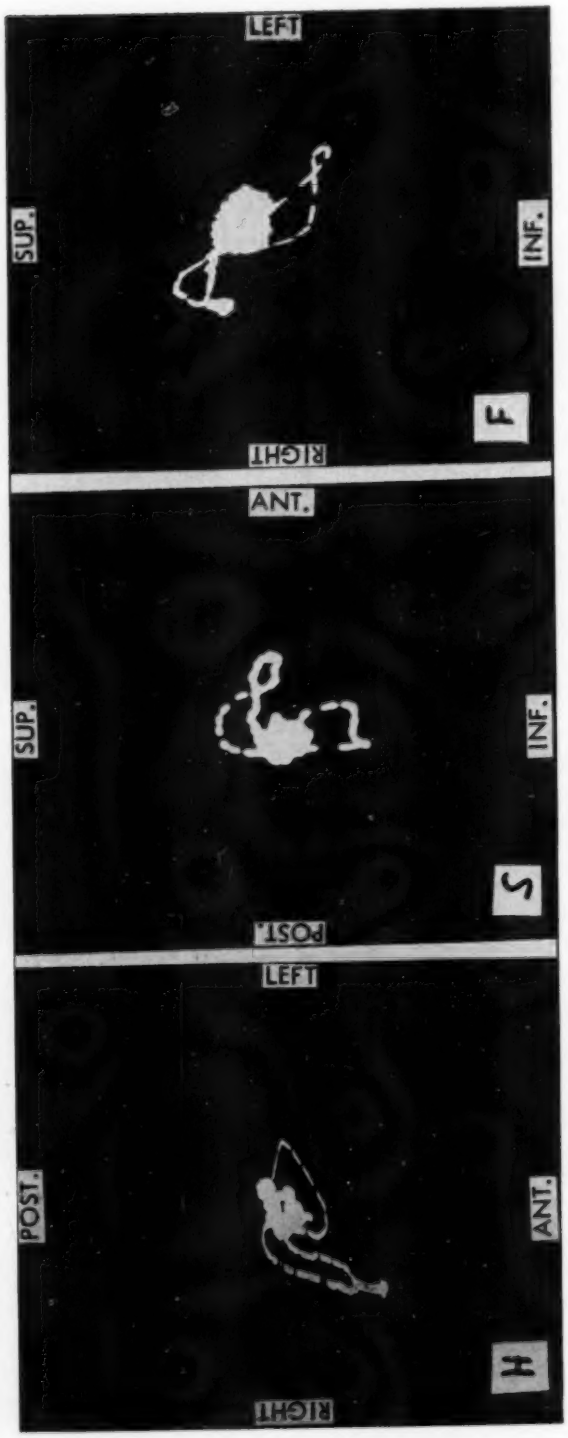
A.

Fig. 3, A.—*(For legend see opposite page.)*



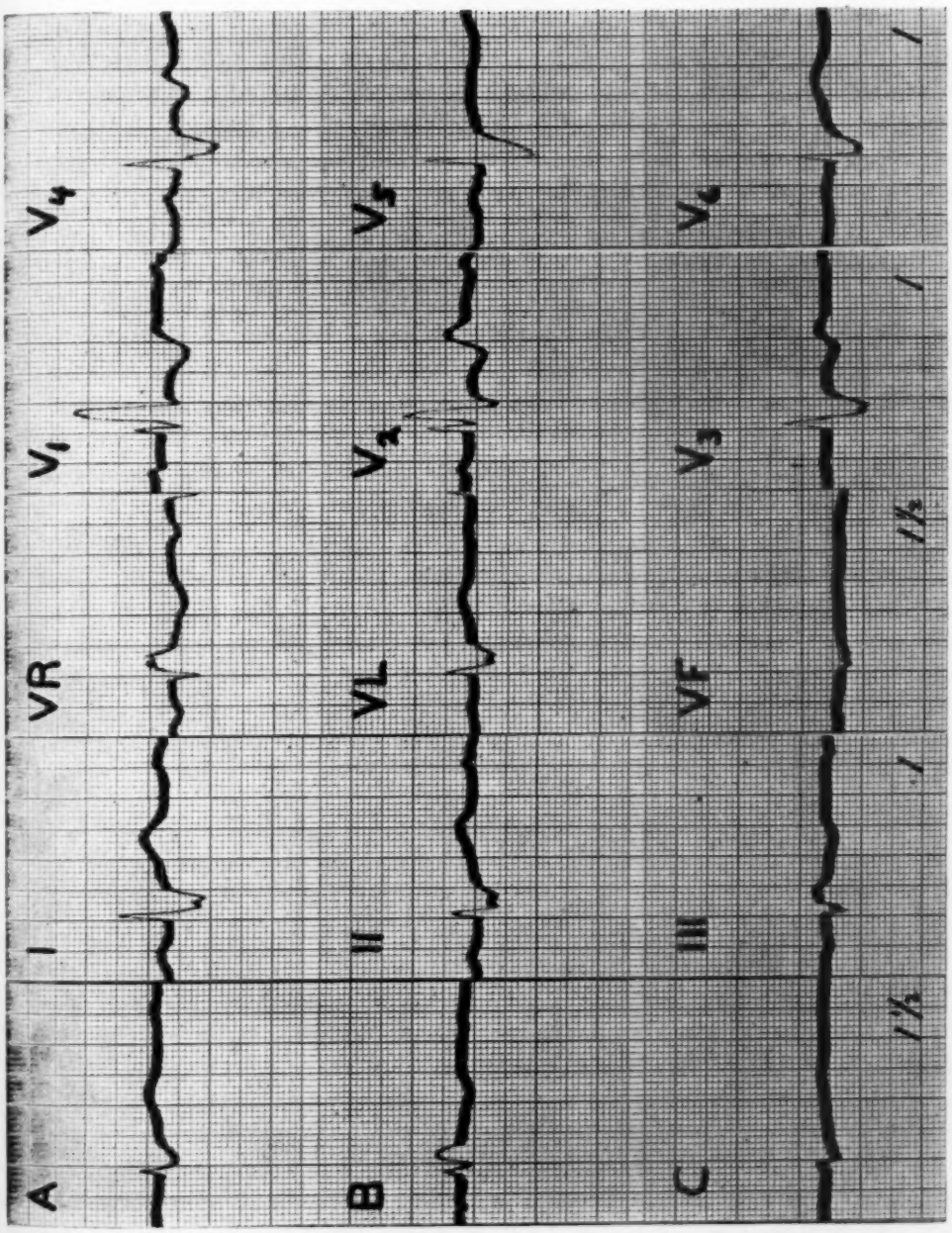
B.

Fig. 3.—A 4½-year-old boy with uncomplicated interventricular septal defect. The vectorcardiogram (*A*) reveals a normal balance of electrical forces with a terminal appendage, oriented to the right and superiorly, and inscribed at a normal speed. This normal variant produces the R' in the electrocardiographic lead V_1 (*B*).



A.

Fig. 4, A.—(For legend see opposite page.)



B.

Fig. 4.—A 6-year-old boy with uncomplicated interventricular septal defect. The vectorcardiogram (*A*) is characteristic of right bundle branch block with a terminal appendage slowly inscribed and oriented to the right, anteriorly and superiorly. The electrocardiogram (*B*) taken at twice the standard paper speed shows an RSR' in Lead V_1 with a QRS duration of 0.11 sec.

Several observers[13,14,43] have commented that right-axis deviation is common, but no axis deviation[13] and left-axis deviation[36] have also been reported. Our findings are essentially in agreement, since six of the nine patients presented with right-axis deviation, two with no axis deviation, and one with left-axis deviation.

Because of the pulmonary hypertension approximating systemic levels, the right ventricle assumes a heavier burden than it does in uncomplicated interventricular septal defect. Bond[44] has reviewed the anatomic features in eighteen cases, all of whom presented anatomic right ventricular hypertrophy while fourteen had associated anatomic left ventricular hypertrophy.

Both the electrocardiograms and vectorcardiograms reflected this increased right ventricular load. With both techniques, seven of the nine patients showed right ventricular hypertrophy. However, occasionally the electrical forces of the left ventricle predominated as evidenced by two patients whose vectorcardiograms indicated left ventricular hypertrophy.

Idiopathic Dilatation of the Pulmonary Artery.—Greene and collaborators[45] reported that three of their four patients with idiopathic dilatation of the pulmonary artery had normal electrocardiograms while the remaining one presented with right-axis deviation and right bundle branch block.

Wenger and Wick,[24] however, found that only one of their three patients with idiopathic dilatation of the pulmonary artery had a normal vectorcardiogram, the other two showed right ventricular hypertrophy. The latter findings are surprising since this malformation has no serious hemodynamic consequences. All four of our patients with this anomaly had normal spatial vectorcardiograms. The electrocardiograms were normal in two patients, while another had an RSR′ in V_1 with a QRS of 0.07 sec. duration, while the fourth patient had the pattern of so-called incomplete right bundle branch block.

COMMENT

In attempting to accurately diagnose the specific nature of a congenital cardiac malformation, the clinician may first classify the patient into the cyanotic or acyanotic groups. Similarly, he may further narrow the diagnostic possibilities by determining the type of ventricular hypertrophy that exists.

In those cases in which the strict electrocardiographic criteria proposed are satisfied[1,4] the type of ventricular hypertrophy present may be established with reliability. Unfortunately, however, the electrocardiogram is not diagnostic in all cases of unilateral ventricular hypertrophy; the vectorcardiogram, on the other hand, offers a greater diagnostic yield. Thus, in patients with normal electrocardiograms the vectorcardiogram may clearly indicate the presence of right or left ventriculary hypertrophy.[2] Patients whose electrocardiograms reveal an RSR′ pattern over the right precordium may easily be separated by the vectorcardiogram into three groups: (1) those with right ventricular hypertrophy, (2) those with normal conduction and a normal balance of electrical forces (Fig. 3), and (3) those with right bundle branch block (Fig. 4).

It should be emphasized that when the electrocardiogram and/or the vectorcardiogram clearly indicate hypertrophy of one ventricle, this does not con-

clusively rule out the presence of some coexisting anatomic hypertrophy of the other ventricle; these techniques merely indicate the resultant of the electrical forces produced by both chambers. The vectorcardiogram in patients with lesions producing biventricular hypertrophy is nonetheless helpful. Most of the patients with Eisenmenger's complex showed right ventricular hypertrophy, while those with uncomplicated interventricular septal defects and patent ductus arteriosus had a normal balance of electrical forces. The finding of right ventricular hypertrophy in the vectorcardiogram in our clinical experience militates strongly against the diagnosis of uncomplicated interventricular septal defect or patent ductus arteriosus.

We have found no specific electrocardiographic or vectorcardiographic pattern associated with any particular lesion. However, there were two findings of possible differential diagnostic value: (1) the patients with interatrial septal defect had the greatest percentage of RSR′ patterns in right precordial electrocardiographic leads. (2) All but two of the patients with the tetralogy of Fallot had vectorcardiograms of the Group 2 type of right ventricular hypertrophy. It has been suggested[46,47] that the malformations producing right ventricular hypertrophy and the severity of the latter may be distinguished by characteristic precordial lead patterns designated as the "surcharge," "adaptation," and "barage" types. We could find no such correlation.

SUMMARY

1. The electrocardiograms and spatial vectorcardiograms of 135 patients with congenital heart disease, in whom the diagnosis was well established, were analyzed. Our findings were compared with those of other authors.

2. No pathognomonic electrocardiographic or vectorcardiographic pattern is associated with any particular anatomic lesion.

3. Both the electrocardiogram and the vectorcardiogram, but particularly the latter, are of considerable aid in the differential diagnosis of the various malformations by indicating the dominant type of ventricular hypertrophy present.

4. In general, patients with the tetralogy of Fallot, pulmonic stenosis, interatrial septal defect, and Eisenmenger's complex show right ventricular hypertrophy; those with tricuspid atresia, subaortic or aortic stenosis, and coarctation of the aorta show left ventricular hypertrophy, while those with uncomplicated patent ductus arteriosus, interventricular septal defect, and idiopathic dilatation of the pulmonary artery show a normal balance of electrical forces.

5. The determination of electrical axis from the standard electrocardiographic leads is found to be of little value in determining the type of ventricular hypertrophy present.

SUMMARIO IN INTERLINGUA

Esseva analysate le electrocardiogrammas e vectocardiogrammas spatial ab 135 patientes con varie congenite malformationes cardiac. Le constatationes assi obtenite esseva comparate con illos obtenite per altere autores. Esseva

trovate nulle configuration electro- o vectocardiographic de valor pathognomonic pro ulle typo de lesion anatomic. Tanto le electrocardiogramma como etiam le vectocardiogramma—sed specialmente iste ultime—se monstrava considerabilemente utile in le diagnose differential de varie malformationes per identificar le typo predominante de hypertrophia presente. In general, patientes con tetralogia de Fallot, stenosis pulmonic, defecto septal interatrial, e complexo de Eisenmenger monstrava hypertrophia dexteroventricular; patientes con atresia tricuspide, stenosis subaortic o aortic, e coarctation del aorta monstrava hypertrophia sinistroventricular, durante que illes con non-complicate patente ducto arteriose, defecto septal interventricular, e dilatation idiopathic del arteria pulmonar habeva un equilibrio normal del fortias electric. Le determination del axe electric ab le electroderivationes standard se monstrava de pauc valor in identificar le typo de hypertrophia ventricular presente.

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VECTOR ANALYSIS OF THE T-DEFLECTION OF THE ELECTROCARDIOGRAM

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THE increasing interest in vectorcardiography may soon lead to a satisfactory method of recording the spatial vectorcardiogram. When this objective has been attained, the method of leading will involve a remote electrode system or its equivalent in which the rectangular components $\vec{E}_x$, $\vec{E}_y$, and $\vec{E}_z$ of the heart's field are available in a minimum of three leads. It appears that identical requirements exist for the leads by which an integrating computer may be expected to determine the coordinates of the mean electrical axis of QRS and of T. Moreover, it is possible that the importance of the integrating circuit may exceed that of the vectorcardiogram.

More than two decades have passed since Wilson and associates[1] described briefly what they felt to be the significance of the areas of the ventricular deflections in the limb leads. Johnston and associates commenced a study of electrical integration of the electrocardiogram at that time and published the results in 1950.[2] At the present time the author is aware of one laboratory[3] other than his own which is now engaged in the development of an electronic integrating computer for the electrocardiogram. In anticipation of the kind of information which may be expected to appear from such computers it seems desirable to elaborate upon the original concept of Wilson and associates[1] with a view toward the adoption of certain conventions which are presented in this article and which may clarify the general concept and serve as a framework for future presentations.

EXCITATION UPON ANY LOCAL PATH

Let us consider excitation along any local path $S' = (e \ . \ . \ . \ f)$ along which accession moves from the point ρ of stimulation anywhere on S', Fig. 1(*a*). The regression process, in terms of its contribution from n paths of the kind S' to the area of the T deflections is

$$\text{Area of } T = \sum_{i=1}^{n} \vec{R}_i \cdot \vec{r}_i/r_i^3$$

$$= \sum_{i=1}^{n} \left[(m+1)\,\vec{e\rho} + (m-1)\,\vec{\rho f} \right]_i \cdot \vec{r}/r_i^3 \qquad (1)$$

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wherein

$$m = \frac{1}{A_1}\left[T_e - T_f\right] = G'/A_1 \tag{2}$$

Here, $\vec{r}$ is the radius vector, large in comparison with $\vec{R}$ from the mid-point of the vector-moment [in square brackets of Eq. (1)] to the field point where the potential is measured. Equation (1) indicates that the regression electromotive force on any path S′ is defined by the vector sum of two electromotive forces. The first of these, on the right of Eq. (1) in square brackets, defines the electromotive force produced by regression against the local gradient $\vec{G}'$. The second term in the square brackets defines the electromotive force produced by regression with or against the local gradient according as the value of m is less or greater than unity. The vector $e\rho$ is the directed line drawn from e to ρ, and the vector $\vec{\rho f}$ is the directed line drawn from ρ to f, Fig. 1(*a*). By equation (2) we observe that the ratio G'/A_1 is directly proportional to the magnitude of the local gradient $\vec{G}'$ and inversely proportional to the magnitude of the accession effect $\vec{A}$ over the local path S′. Also, T_e and T_f are the durations of the excited state at the end points of S′. Let us consider all paths S′ along which excitation ordinarily passes. We may take e as a point on the endocardial surface and f as a point on the epicardial surface. For all such paths S′ we have a set of points *e* on the endocardial surface and a second set *f* on the epicardial surface. The two sets *e* and *f* now include every point upon the surface of the ventricular muscle mass. Moreover, we may ordinarily regard the point ρ of stimulation as a set of points at the set *e* whence all the vectors of the first term vanish since $e\rho = 0$. Then equation (1) reduces to

$$\text{Area of } T = \sum_{i=1}^{n}\left[(m-1)\,\vec{\rho f}\right]_i \cdot \vec{r}_i/r_i^3 \tag{3}$$

and m is now defined by

$$m = \frac{1}{\rho f}\left[T_\rho - T_f\right] = G'/A \tag{4}$$

We observe by Eq. (3) that if $m > 1$ the contribution to the area of the T deflection produced by regression $\vec{R}$ upon the local path S′ is positive, and if $m \leq 1$ this contribution is negative.* Moreover, the local accession effect is $\vec{\rho f}$, and if $m = 1$, the contribution to the area of T is zero and, by the value of m, we have $\vec{\rho f} = \vec{G}'$ that is, the accession contribution is a vector equal in magnitude and direction to the local gradient over S′ = (e . . . f).

We further observe that the gradient $\vec{G}'$ is a summation of the variations, $-\nabla T_o$, over the entire path S′ and is equal in magnitude to the difference in the duration of the excited state T_o at e denoted by T_e and its duration at f denoted by T_f. The direction of $\vec{G}'$ is that of a line from e where the duration of T_o is greatest toward or through f where the duration of T_o is least. Upon

*Apart from cosine of the angle $(\vec{R}, \vec{r})$.

this line the variations $-\nabla T_o$ are greatest in the direction of T_o decreasing. It is known that both accession and regression are lamellar[4] and that the potential depends upon the order of accession and the order or regression of the surface units only of the muscle mass. Consequently, the only durations of the excited state which can affect the form of T are those upon the ventricular surfaces, that is at the set of points *e* and at the set *f*. The order of regression

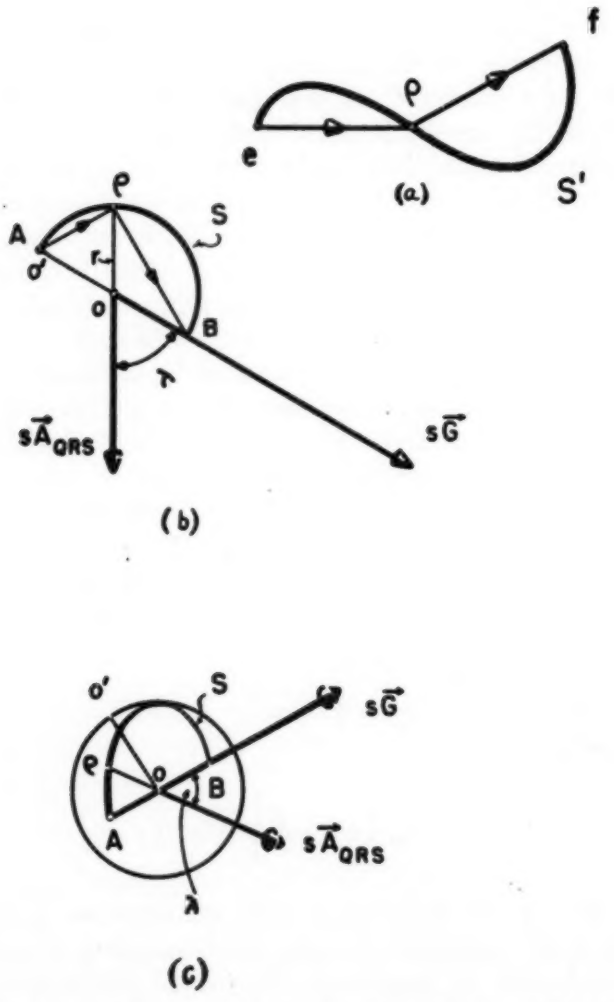


Fig. 1.—(*a*), Arbitrary path S′ = (e . . . f) along which accession moves from the arbitrary point ρ of stimulation. (*b*), Space vectors $s\vec{A}_{QRS}$ and $s\vec{G}$ in relation to the axis S of the average ventricular fiber. Stimulation is arbitrary at ρ. Regression is defined in terms of the vectors $\vec{A}_\rho$ and $\rho\vec{B}$, see text. (*c*), The average ventricular fiber S varies over the surface of a sphere of radius $r = sA_{QRS}/2$ about O, and ρ is any point on the sphere. OO′ identifies the axis of the sphere, see text.

upon all the paths S′ between these surfaces is immaterial. Thus the gradient must be considered uniform over every path S′ along which accession moves.

While these considerations lead to an interpretation of the area of T in terms of its primary changes, $\vec{G}'$ and secondary changes $-\vec{A}$, no direct infor-

mation is obtained upon the local duration T_o of the excited state for the set of points *e* and the set *f* upon the ventricular surfaces.

If the ventricular surface point chosen for exploration is one which permits the excitation wave to both approach and pass the point, the intrinsic deflection crosses the base line at the time at which the membrane current in the fiber at the point reverses itself, that is when the accession dipole is beneath the electrode tip. In this way the onset of T_o at that point is measured.[5] However, as Wilson and associates have pointed out[6] there is no corresponding point upon the regression deflections relative to which the end of T_o at that point can be measured. Many years ago Wilson and Herrmann[7] attempted estimations of T_o relative to the refractory period in dog and concluded that ". . . under normal conditions, the duration of the state of excitation is approximately uniform throughout the ventricular muscle."

EXCITATION UPON THE AVERAGE VENTRICULAR FIBER

If instead of the space curve $S' = (e \ . \ . \ . \ f)$ we consider the space curve $S = (A \ . \ . \ . \ B)$ which describes the axis of the (fictitious) average ventricular fiber along which accession moves from the point (or centroid) ρ of stimulation, and if the summations are taken over the QRS and T intervals of three mutually perpendicular leads based upon a remote electrode system which is suitable for recording the $\vec{E}_x$, $\vec{E}_y$, and $\vec{E}_z$ components of the heart's field, we have the net areas for the coordinates of the space vectors $s\vec{A}_{QRS}$ and $s\vec{A}_T$. Moreover, the latter is defined by the relation

$$\int_T^S \left\{ \vec{E}_x + \vec{E}_y + \vec{E}_z \right\} dt \sim$$

$$s\vec{A}_T = (n + 1)\vec{A\rho} + (n - 1)\vec{\rho B} \tag{5}$$

It will be convenient to always inscribe S as a semicircular arc of radius $r = sA_{QRS}/2$ centered upon the origin O of the ventricular gradient $s\vec{G}$, in the plane defined by $s\vec{A}_{QRS}$ and $s\vec{G}$ and always upon the opposite side of $s\vec{G}$ from the vector $s\vec{A}_{QRS}$, Fig. 1(*b*). If $\vec{g}$ is a unit vector in the direction of $s\vec{G}$, the point A of S is at the origin of the vector $(r + sG)\vec{g}$, and the point B is on $s\vec{G}$ at a distance r from O, the origin of both $s\vec{A}_{QRS}$ and $s\vec{G}$. The point ρ of stimulation always lies upon S at the end of the line $\vec{O\rho}$ in the direction of $-s\vec{A}_{QRS}$. Moreover, the scalars $A\rho$ and ρB are defined by

$$\left.\begin{aligned} A\rho &= sA_{QRS} \sin \lambda/2 \\ \rho B &= sA_{QRS} \cos \lambda/2 \end{aligned}\right\} \tag{6}$$

wherein λ is the angle made by $s\vec{A}_{QRS}$ and $s\vec{G}$. In equation (5) n is a positive number which takes on the value zero when the gradient is zero as may be observed from the relation

$$n = \frac{1}{2r}\left[T_A - T_B\right] = sG/sA_{QRS} \ . \tag{7}$$

A substitution in Eq. (5) which replaces n by its identity on the right hand side of Eq. (7) gives Wilson's basic electrocardiographic equation

$$\vec{sG} = sA\vec{_{QRS}} + sA\vec{_T} \quad . \tag{8}$$

The form Eq. (5) will be found more convenient than Eq. (8) for analytical purposes particularly when used with a model similar to that of Fig. 1(*b*).

SECONDARY T-WAVE CHANGES

The position of ρ on S describes all secondary T-wave changes. The normal radius r of arc S is fixed by normal values of $sA\vec{_{QRS}}$ and, if ρ is on S of an arc inscribed by r increased, there is hypertrophy of the average element of ventricular muscle. When the enlarged arc S is anterior to O, $sA\vec{_{QRS}}$ is directed posteriorly, and hypertrophy of the posterior (left) ventricle is indicated. When the enlarged arc S is posterior to O, $sA\vec{_{QRS}}$ is directed anteriorly, and hypertrophy of the anterior (right) ventricle is indicated. When the direction of $\vec{sG}$ is normal ρ varies on S over the normal interval $0 \leqq A\rho \leqq r$, or $\lambda \leqq \pi/3$ which indicates that normally the centroid of stimulation ρ does not necessarily coincide with the region A where the average duration T_o of the excited state is maximum. The normal variations of n are in the interval $1.5 \leqq n \leqq 2.0$. If we take $\lambda = \pi/3$ and $n = 2$ we find the maximum normal value of the angle $(sA\vec{_{QRS}}, sA\vec{_T}) = 90°$. With the direction of $\vec{sG}$ normal, if ρ on S exceeds the distance $A\rho = r$, the manner of stimulation of the ventricles is abnormal. Examples are incomplete and complete right and left bundle branch block or interventricular block of other types. If the block is uncomplicated by primary T-wave changes $\vec{sG}$ is normal and r is increased. When the shift of ρ on S is anterior to O the block is posterior or left. When the shift of ρ on S is posterior to O, the block is anterior or right. In pre-excitation ρ shifts posterior and to the right on S in one type and posterior and to the left in the other type.

In general, the first term on the right in Eq. (5) indicates the electromotive force produced by regression moving against the ventricular gradient, and the last term indicates the electromotive force produced by regression moving with or against the ventricular gradient according as the value of n is less or greater than unity.

Inasmuch as abnormal $sA\vec{_{QRS}}$ and $\vec{sG}$ may have any direction in space, the variations of S are over the surface of a sphere of radius r about O, and those of ρ are any point on the sphere, Fig. (1*c*). Obviously, when the direction of $\vec{sG}$ is abnormal, the normal and abnormal variations of ρ are with respect to the pole O′ of the sphere of radius r about O. It might appear that the value of $sA\vec{_T}$ is not defined by Eq. (5) for $sA\vec{_{QRS}}$ approaching zero where by Eq. (7) it appears that $n \rightarrow \infty$. This is not the case, however, for

$$\begin{aligned} \underset{sA_{QRS} \rightarrow 0}{\text{Lim}} [sA\vec{_T}] &= n(\vec{A\rho} + \vec{\rho B}) - (\vec{\rho A} + \vec{\rho B}) \\ &= \left(\frac{sG}{sA_{QRS}}\right) sA_{QRS}\,\vec{g} - sA\vec{_{QRS}} \\ &= \vec{sG} - 0 \end{aligned}$$

which obviously agrees with Eq. (8).

PRIMARY T-WAVE CHANGES

According to equation (5) n is directly proportional to the magnitude sG of the ventricular gradient which in turn depends upon the average duration T_o of the excited state at the region A denoted by T_A and its duration at B denoted by T_B. The point A may ordinarily be taken as a region on the endocardial surface where the average value of T_o is greatest, and B may be taken as a region on the epicardial surface where the average value of T_o is least. The magnitude of $\overrightarrow{sG}$ is a measure of the difference in the average durations T_A and T_B, and the direction of $\overrightarrow{sG}$ is that of a line along which the average variations are greatest. When $\overrightarrow{sA_{QRS}}$ is normal, the normal value of $n = 2$ indicates that the difference in the durations $[T_A - T_B]$ is ordinarily twice as long as is required for accession to pass from A to B on S. It is well known that cooling[8] prolongs the values of T_o locally and elements of S at A appear to have a blood-cooled "radiator system" involving the lungs. On the other hand, the heat of ventricular contraction is more poorly dissipated from the epicardial surface. Our choice of normal A and B are therefore reasonable. In addition, the pressure gradient across the ventricular wall alters the environment of elements of S at A in comparison with those at B.

If the duration of the excited state at A is equal to that at B, $\overrightarrow{sG} = 0$ and $n = 0$ and equation (5) gives

$$\overrightarrow{sA_T} = \overrightarrow{A\rho} - \overrightarrow{\rho B} = -(\overrightarrow{\rho A} + \overrightarrow{\rho B}) = -\overrightarrow{sA_{QRS}} \tag{9}$$

which indicates that $\overrightarrow{sA_T}$ is equal in magnitude and opposite in sign to $\overrightarrow{sA_{QRS}}$; or, the net area of T in the component leads is equal in magnitude and opposite in sign to the corresponding net area of QRS.

If in Eq. (5) $\lambda = 0$, ρ is at A, a normal position when r is not increased and the direction of $\overrightarrow{sG}$ is normal. Then $A\rho$ is zero, and the vector $\overrightarrow{A\rho}$ vanishes and Eq. (5) gives

$$\overrightarrow{sA_T} = (n - 1)\ \overrightarrow{\rho B} \tag{10}$$

If in addition $n = 1$ equation (10) gives

$$\overrightarrow{sA_T} = 0$$

and therefore by the value of n we have

$$\overrightarrow{sA_{QRS}} = \overrightarrow{sG} \ . \tag{11}$$

The electrocardiogram in Fig. 2*a* is of this kind, and the approximate diagram is shown in Fig. 2*b*. It may be noted that the unipolar precordial leads to the left of transition indicate a semilocal gradient wherein the net area of T is not zero, and Eq. (10) cannot properly be "differentiated" with respect to time for

a local gradient. When $n = 1$ either T_A is diminished (short Q-T interval) or T_B is increased (normal Q-T interval). The legend of Fig. 2*a* may be consulted for clinical details.

When $\lambda = \pi$, ρ is at B and Eq, (5) gives

$$s\vec{A}_T = (n + 1)\, \vec{A\rho} \tag{12}$$

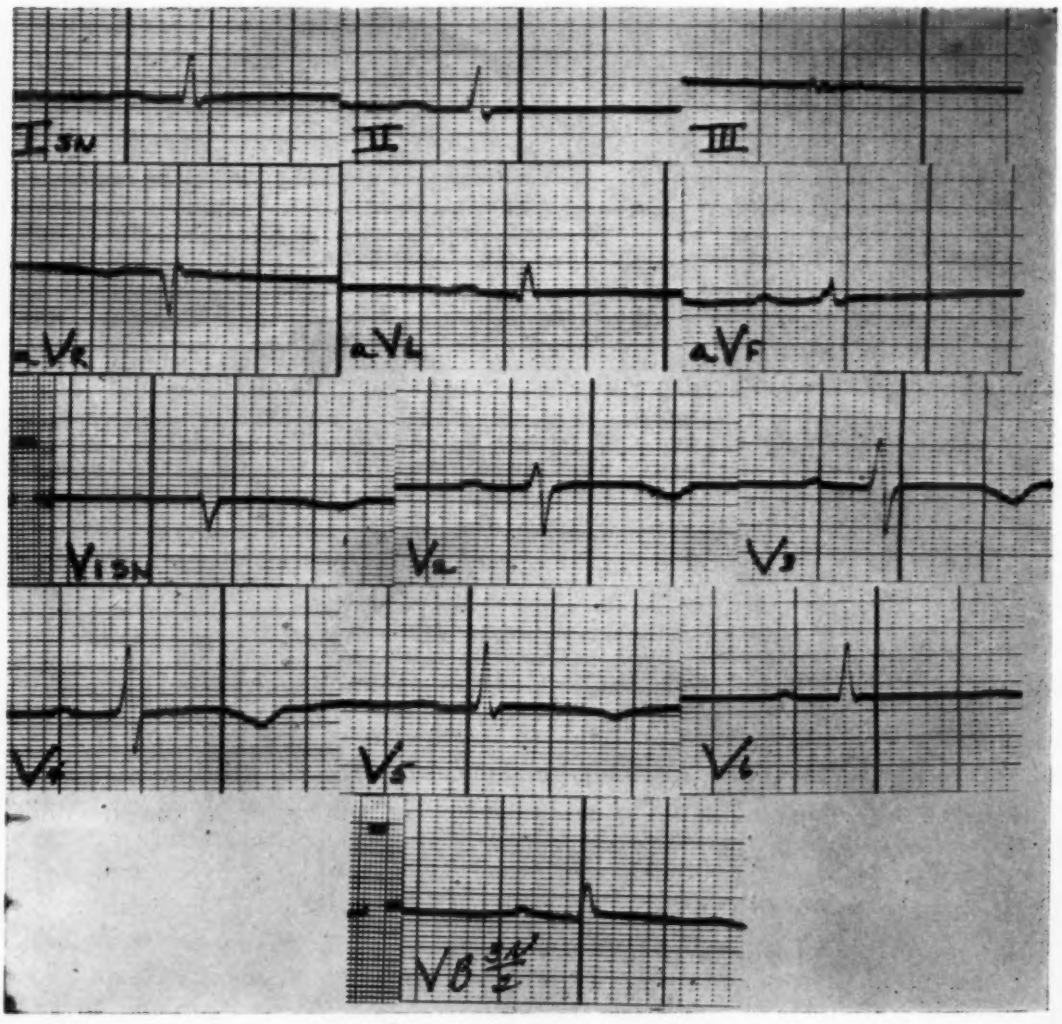
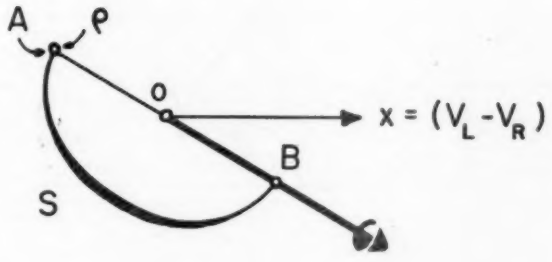


Fig. 2*a*.—The subject is a white female, aged 61 years, who gave a history of "two coronaries" in the past 7 months. There were no symptoms or signs of congestive failure. Discomfort, suggestive of brief attacks of heart pain, was present with no cardiac enlargement. The heart rate was only 60 per minute despite a pallor indicative of mild anemia. The electrocardiographic diagnosis is "primary T-wave changes, non-specific."

which indicates that regression over S is entirely against the gradient, $n \neq 0$. If $\vec{sG}$ is normal in direction, the variation of ρ on S is striking and might result from a ventricular extrasystole starting at the epicardial surface where the

average duration of T_o is normally least. Let n have a normal value of 2, then Eq. (12) gives

$$\begin{aligned} s\vec{A}_T &= 3\ \vec{A\rho} = -3\ \vec{\rho A} \\ &= -3s\vec{A}_{QRS} \end{aligned} \tag{13}$$



$$s\vec{G} = s\vec{A}_{QRS}$$

$$s\vec{A}_T = (n-1)\vec{\rho B},\quad n=1$$

Fig. 2*b* displays the vector diagram. Note that the area of T is not zero in the precordial leads. Diagnostic considerations should include hypothyroidism. The scale is arbitrary. The posterior direction of $s\vec{A}_{QRS}$ is approximated from the sagittal lead (V_B–V_T).

which indicates that $s\vec{A}_T$ is three times the magnitude of $s\vec{A}_{QRS}$ and is in the opposite direction, a striking secondary T-wave change. If instead n = 0 in Eq. (12) we have

$$s\vec{A}_T = \vec{A\rho} = -s\vec{A}_{QRS} \tag{14}$$

which indicates that the area of T is equal and oppositely directed to that of QRS in the component leads. The T-deflection is now much less striking than in Eq. (13) while QRS is unchanged but abnormal. The value of n = 0 indicates a primary T-wave change in addition to the secondary change which resulted from the assumed abnormal manner of stimulation.

The equation

$$s\vec{A}_T = (n+1)\ \vec{A\rho} \tag{12}$$

is not confined to a description of the ventricular extrasystole. The electrocardiogram in Fig. 3,*a* is of the same class wherein regression is entirely against the gradient. However, the gradient is abnormal at −150° in RLF and slightly anterior, while $s\vec{A}_{QRS}$ is normal at +30° in RLF and slightly posterior, Fig. 3*b*. The value of n = 2 is abnormal and Eq. (12) gives

$$s\vec{A}_T = 3\ \vec{A\rho} = -3\ s\vec{A}_{QRS}$$

which is identical to equation (13). The centroid of stimulation is normal at O′ the pole of the sphere of radius r about O and is incidentally at B. T_A is taken as a region on the epicardial surface and T_B a region on the endocardial

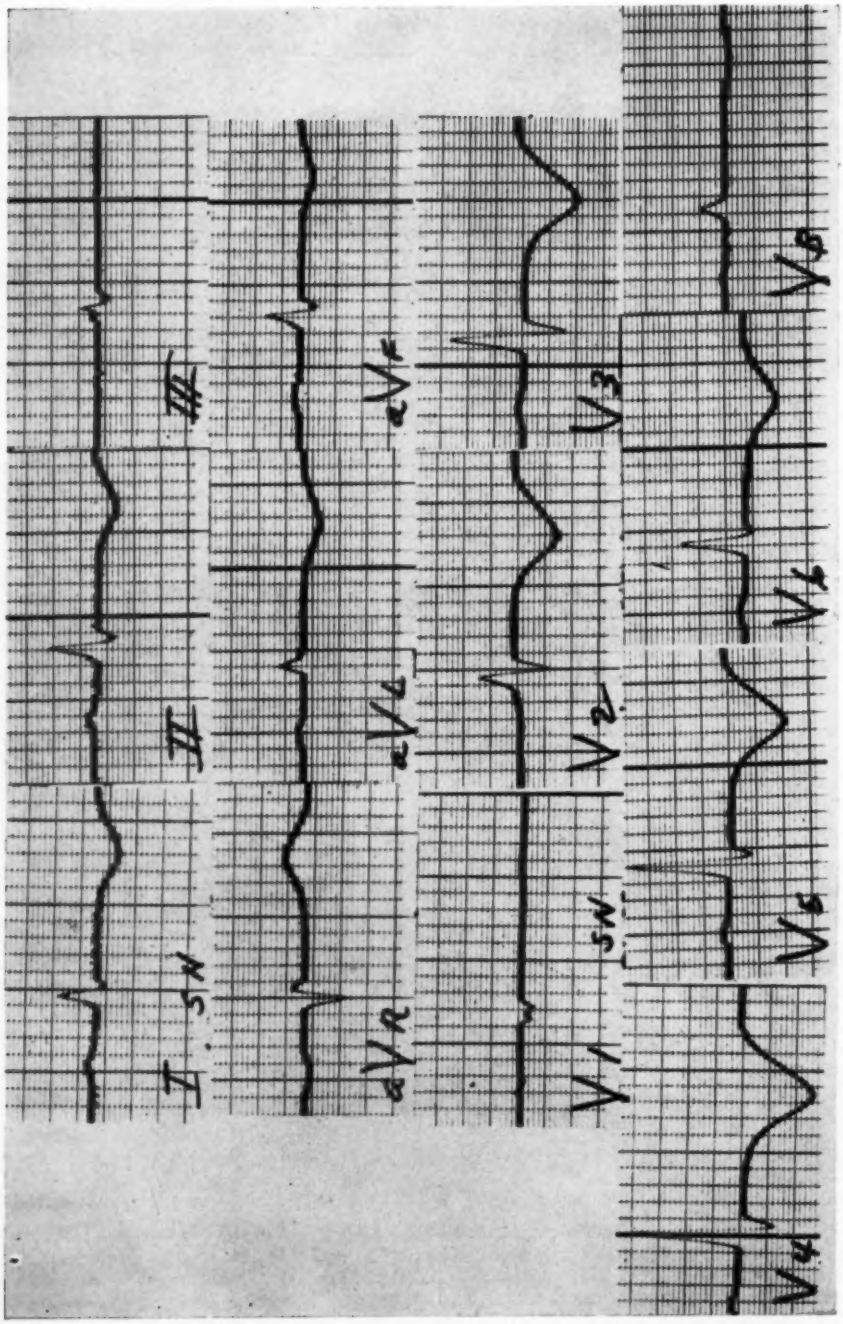


Fig. 3*a*.—The electrocardiographic diagnosis is "primary T-wave changes due to prolonged duration of excited state over the epicardial regions." The associated vector diagram is shown in Fig. 3*b*. The subject, a 73-year-old white female, suffered from attacks of heart pain. When the intensity or frequency of the attacks increases and is associated with appearance of T-wave changes of the kind displayed, impending infarction must be considered.

surface. The duration of the excited state T_o is greatly prolonged over most of the epicardial regions anteriorly and apically. The duration T_B is not greatly reduced for the RS-T interval is not greatly decreased. The T-wave changes are primary and are associated with a clinical picture of bouts of heart pain (angina pectoris). The subject was a white female, aged 73 years, with otherwise normal clinical findings. The total picture makes it likely that the primary T-wave changes are evidence of ventricular ischemia on the basis of degenerative disease of the coronary arteries.

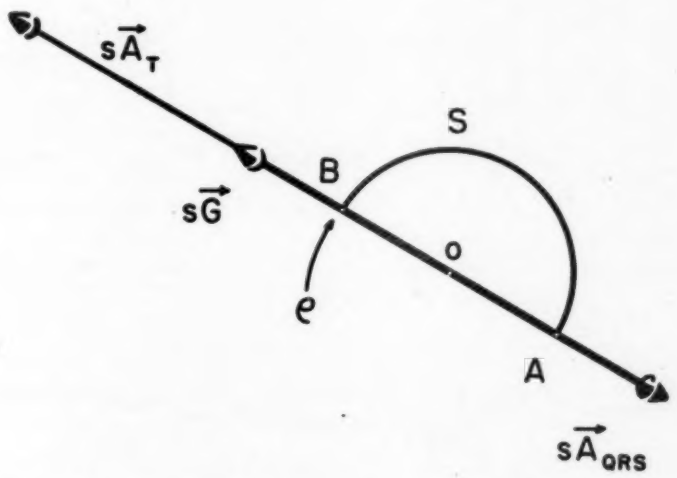


Fig. 3*b*.—Vector diagram of the curve in Fig. 3*a*. The direction of the gradient is abnormal. $s\vec{A}_{QRS}$ is normal and regression is entirely against the gradient, see text.

In equation (5) let λ have a normal value of 0, then ρ on S is at A and Eq. (10) holds. Take $s\vec{A}_{QRS}$ as normal and let n exceed its upper value of 2, say $n = 4$, then Eq. (10) gives

$$s\vec{A}_T = 3\rho\vec{B} = 3\ s\vec{A}_{QRS} \tag{15}$$

which indicates that $s\vec{A}_T$ is three times the magnitude of $s\vec{A}_{QRS}$ and in the same direction, a striking primary T-wave change. The T waves are large and positive in Leads I and II or in Leads II and III. The electrocardiogram in Fig. 4*a* is an approximate example, and Fig. 4*b* is the vector diagram. Either T_B at the epicardial surface is markedly reduced (Q-T interval normal) or T_A at the endocardial surface is actually increased (prolonged Q-T interval). Since the direction of $s\vec{G}$ is normal, T_o may be prolonged equally with respect to the normal durations over the greater area of the endocardial surface. The particular etiologic factor or factors operating must be determined primarily from the details of the clinical picture. The causative factor is seldom suggested by peculiar variations of the wave form of the T deflection (see the legend of Fig. 4*a* for clinical details).

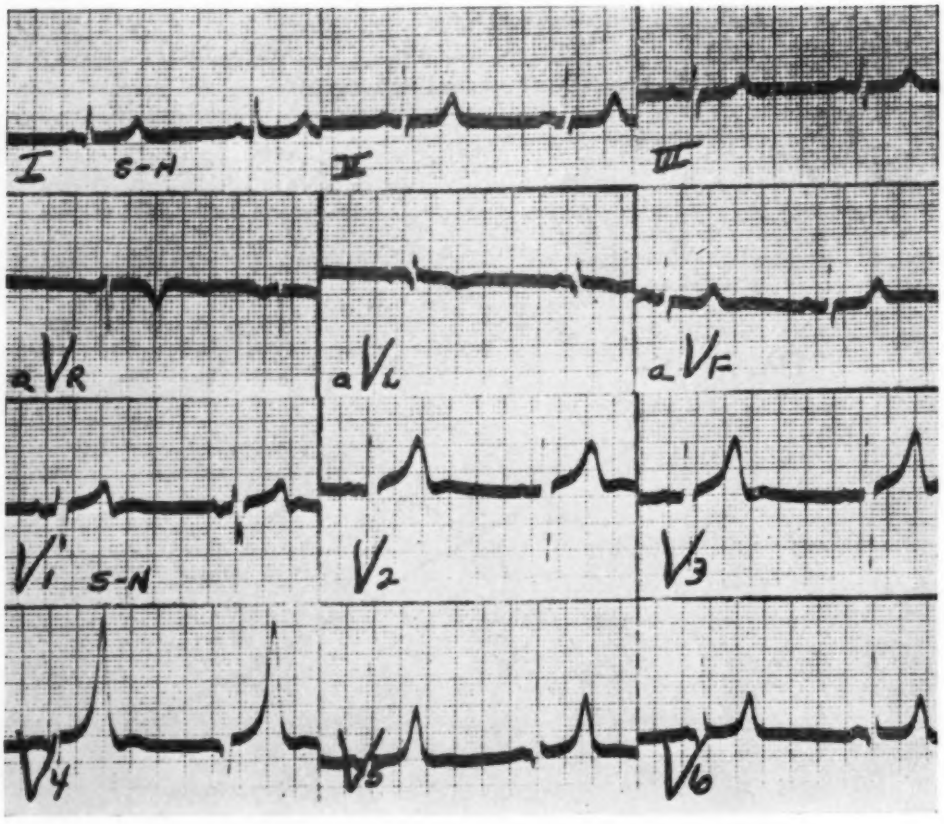


Fig. 4*a*.—The subject, a white male, aged 66 years, gave a history of primary syphilis at age 26 followed by penicillin therapy at age 59. Attacks of heart pain commenced 8 months prior to taking the electrocardiogram. The attacks occurred during the day or night and might last as long as two hours. There was slight cardiac enlargement; B.P., 200/100; no signs of congestive failure. The electrocardiographic diagnosis is "primary T-wave changes, possibly due to prolongation of the excited state over the endocardial regions."

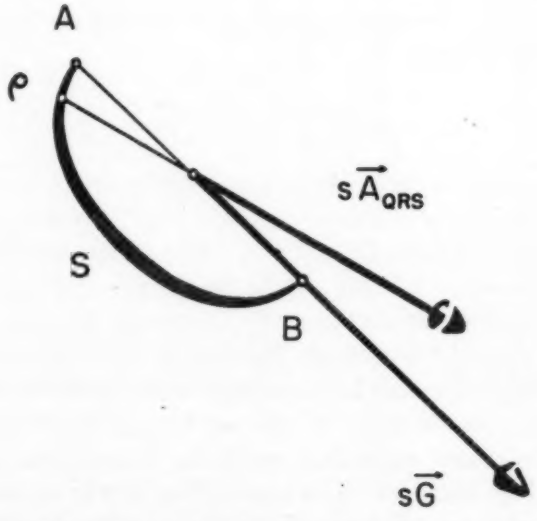


Fig. 4*b*.—Vector diagram of Fig. 4*a*. The suspected etiologic factor is syphilitic aortitis with partial closure of the coronary ostia.

REMARKS

In the foregoing discussion of equation (5) only a few of the more obvious analytical properties have been indicated and all specific values must be considered approximate. Relations (9) through (15) show that the pictorial display of the vector $\overrightarrow{sA_T}$ is unnecessary. When the directions of $\overrightarrow{sA_{QRS}}$ and $\overrightarrow{sG}$ are known together with values λ and n, the T problem is solved as far as the electrocardiogram in remote leads is concerned.

If in Fig. 1*b*, we take $\lambda = 0$, then ρ remains in a normal position at A and sA_T is defined by equation (10). We retain $n = 2$ as in the figure. Whence

$$\overrightarrow{sA_T} = \overrightarrow{\rho B} = \overrightarrow{sA_{QRS}} \quad . \tag{16}$$

We next let T_A have a normal value of 0.36 sec. and suppose that the QRS interval is normal at 0.09 sec. It will be instructive to compute the time after stimulation at which regression is complete at the regions A and B. At the region A this time is 0.36 sec. The duration of the excited state at B is given by

$$\begin{aligned} T_B &= T_A - sG = T_A - n\, sA_{QRS} \\ &= 0.36 - 0.18 = 0.18 \text{ sec.} \end{aligned}$$

Accession takes 0.09 sec. to reach B, whence $(0.09 + T_B) = 0.27$ sec. at which time regression is complete at B. Equation (16) indicates that regression moves from B to A in 0.09 sec. producing the vector $\overrightarrow{\rho B} = \overrightarrow{AB} = \overrightarrow{sA_{QRS}}$. If we add this time to the time after stimulation at which regression is complete at B we have $(0.09 + 0.27) = 0.36$ sec., the time after stimulation at which regression is complete at A. Now, $\overrightarrow{sA_{QRS}}$ and $\overrightarrow{sA_T}$ have positive net areas of equal magnitude in the component Leads I and II. If the initial positive area under T_1 or T_2 is added to the initial positive area under QRS_1 and QRS_2, respectively, for a local gradient in the vicinity of A at the endocardial surface, it may be supposed that local regression near A is responsible for the initial area under T. Actually regression begins at B only 0.09 sec. after the onset of QRS, and the rapid rate at B on the epicardial surface means regression dipoles of large moment in comparison with those at A. Consequently, the initial positive areas of T_1 and T_2 are as much, if not more, due to regression activity near B. On the other hand, the final positive area under T_1 and T_2 is due almost entirely to regression activity near A since regression is complete at B in 0.27 sec. and continues for 0.09 sec. longer at A.

The conditions placed upon the gradient concept by Wilson and associates[1] and elaborated upon here are such that the intervals over which integration occurs for the space vectors $\overrightarrow{sA_{QRS}}$ and $\overrightarrow{sA_T}$ cannot be "differentiated" with respect to time for a local gradient as suggested by others.[9] Points on S within the interval of summation are along the fictitious average ventricular fiber and the end points of S lie upon the ventricular surfaces. If the ventricular gradient is to measure the difference in the average duration of the excited state between the surface region where the average is greatest and surface region where the average is least, it must be independent of the path between these regions where

the order of regression is immaterial, that is, it must be uniform over the average ventricular fiber. It is not possible to anticipate activity upon a local path S′ from the activity upon the average fiber S nor can excitation upon the latter be judged by the excitation upon the former path.

Although the most rapid directional variations of T_o upon the average ventricular fiber are measured by the sum of two electromotive forces $s\vec{A}_{QRS}$ and $s\vec{A}_T$, the gradient $s\vec{G}$ is not itself an existent electromotive force but is the directional derivative of a time interval T_o, a function of position in space, and T_o is the duration of the excited state at any point and is constant at that point for any beat under consideration.

APPENDIX

Let S′ = (e . . . f), Fig. 1*a*, be a uniform path along which accession moves (during a given ventricular excitation) between some end point e ordinarily upon the endocardial surface and a second point f ordinarily upon the epicardial surface. Upon two elements of path dS'_1 and dS'_2 on either side of the point ρ of stimulation somewhere on S′ have defined the accession process, a vector $\vec{dA}$ equal to the product of the mean electromotive force by a time interval. The recovery process (regression) is also identified by a vector $\vec{dR}$ equal to the product of a (smaller) mean electromotive force by a (longer) time interval. Thus with a similar environment for each element of the path we have

$$\vec{dA} = -\vec{dS}'_1 + \vec{dS}' \tag{1}$$

and

$$\vec{dR} = \vec{dS}'_1 - \vec{dS}'_2 \quad . \tag{2}$$

The stipulation of an identical environment for each element of path is unphysiological, and the result $\vec{dA} + \vec{dR} = 0$ for the total electrical effect $\vec{dG}'$ of excitation can hardly be considered reasonable. In fact, observation indicates that a large number of factors which determine the internal and external environment of the myocardial fibers along S′ may produce variations in T_o, the duration of the excited state of a given element which is almost if not entirely due to variations in dR the magnitude or length of the regression process. In any event we may consider

$$T_o = -f(G'^n) \tag{3}$$

and

$$\vec{dG}' = \vec{dA} + \vec{dR} \quad . \tag{4}$$

Further, we may solve Eq. (3) for the most rapid decrease of T_o in any direction. The result is

$$-\nabla T_o = f'(G'^n)\, nG'^{n-1} \frac{\vec{G}'}{G'} \quad . \tag{5}$$

And evidently $-\nabla T_o$ is a vector parallel to $\vec{G}'$. In particular, if T_o is taken as the magnitude of $\vec{G}'$, $f'(G'^n)$ in Eq. (5) is unity and $n = 1$; also $\vec{G}'/G'$ is a unit vector $\vec{U}$ in the direction of $\vec{G}'$. Moreover, the change in T_o upon the element of path dS′ is given by

$$\vec{U} \cdot dS' = -\nabla T_o \cdot \vec{dS}' \tag{6}$$

which may be written

$$[\vec{U} + \nabla T_o] \cdot \vec{dS}' = 0 \tag{7}$$

and since $\vec{dS}'$ is a vector in any direction the vector $[\vec{U} + \nabla T_o]$ has no compound in any direction and

$$\vec{U} = -\nabla T_o \quad . \tag{8}$$

It will be convenient to define the magnitude of $\vec{dG}'$ in terms of the time interval required by accession to move over the elements $\vec{dS}'_1$ and $\vec{dS}'_2$. To this end we let m denote the scalar such that $m \equiv \frac{dG'}{dA_1}$. Then $\vec{dG} = m\vec{dA}_1$ and we have

$$\vec{dG}' = m(\vec{dS}_1 + \vec{dS}_2) \quad . \tag{9}$$

Here, $\vec{dA}_1 = 2\vec{dS}_1 + \vec{dA}$, and $dA_1 = dA$ when ρ is at e or when the points e, ρ, f define a right triangle with ρ at the apex of 90° and the path being any path beginning at e passing through ρ and terminating at f, and according to equations (4) and (9) the regression process is now defined by

$$\vec{dR} = m(\vec{dS}'_1 + \vec{dS}'_2) - \vec{dA} \tag{10}$$

which, according to equation (1) may be written

$$\vec{dR} = (m + 1)\ \vec{dS}'_1 + (m - 1)\ \vec{dS}_2 \quad . \tag{11}$$

Now, the total electrical effect produced by regression upon $S' = (e \ldots f)$ is obtained by forming the line integral of $\vec{dR}$ along S′ upon either side of ρ the point of stimulation. Thus

$$R = \int_{\rho}^{e} (m + 1)\ \vec{dS}'_1 + \int_{f}^{\rho} (m - 1)\ \vec{dS}'_2 \quad . \tag{12}$$

In this relation m is not constant during integration, and it will be convenient to determine the summations for dG'/dA_1 separately. Integrating Eq. (12) we get

$$\vec{R} = (m + 1)\ \vec{e\rho} + (m - 1)\ \vec{\rho f} \quad . \tag{13}$$

By Eq. (1) we may integrate accession over the path.

Thus

$$\begin{aligned} \vec{A}_1 &= 2\int_{\rho}^{e} \vec{dS}'_1 - \int_{\rho}^{e} \vec{dS}'_1 + \int_{f}^{\rho} \vec{dS}'_2 \\ &= 2\,\vec{e\rho} + \vec{\rho e} + \vec{\rho f} \\ &= 2\,\vec{e\rho} + \vec{A} \end{aligned} \tag{14}$$

and, by equations (6) and (14) we have

$$\begin{aligned} m &= 1/A_1 \int \vec{U} \cdot \vec{dS}' = 1/A_1 \int_{f}^{e} \nabla T_o \cdot \vec{dS}' \\ &= 1/A_1\ [T_e - T_f] = G'/A_1 \quad . \end{aligned} \tag{15}$$

The dependency of the potential or its derivative upon a closed curve or an aggregation of closed curves inscribed upon the ventricular surfaces,[4] epicardial and endocardial, indicates that the variations $-\nabla T_o$ upon the path between these surfaces, like the order of accession and regression, is immaterial. In addition, the variations $-\nabla T_o$ are independent of ρ the point of stimulation.

A set of points *e* and a set *f* will include all the points upon the ventricular surfaces, and between the sets there will be n paths of the kind S′ which constitute the total electrical effect of excitation. The contribution to the area of T at any point p by excitation upon a local path is described by an equation of the form (13) except for the orientation of the path S′ with respect to the point p of observation. The effect of the electromotive force $\vec{R}$ varies inversely with the square of the distance r of p from the midpoint of $\vec{R}$ (large in comparison with $\vec{R}$) and directly with the cosine of the angle $(\vec{r}, \vec{R})$. Consequently the area of T at p is given by

$$\int_T^S V_P \, dt = \sum_{i=1}^{n} \vec{R}_i \cdot \vec{r}_i/r_i^3$$

$$= \sum_{i=1}^{n} [(m + 1) \, \overrightarrow{e\rho} + (m - 1) \, \overrightarrow{\rho f}]_i \cdot \vec{r}_i/r_i^3 \qquad (16)$$

If upon each of the n paths ρ is at e all vectors of the form $\overrightarrow{e\rho}$ vanish and, remembering $\vec{A}_1 = \vec{A}$, Eq. (15) reduces to

$$\int_T^S V_P \, dt = \sum_{i=1}^{n} \vec{R}_i \cdot \vec{r}_i/r_i^3$$

$$= \sum_{i=1}^{n} [(m - 1) \, \vec{A}]_i \cdot \vec{r}_i/r_i^3 \qquad (17)$$

If $\vec{r}$ is not large in comparison with $\vec{R}$, equations (16) and (17) are unsuitable. However, take ρ at e then if r_1 is the distance from f to p and r_2 the distance from e to p we have $\vec{M} =$

$$\text{Lim } (\Delta\vec{R}/\Delta v), \quad \Delta v \to 0$$

$$\int_T^S V_P \, dt = \iiint \frac{\vec{M} \cdot \vec{r}}{r^3} \, dv = \iint \frac{(m - 1) \, (r_2 - r_1)}{r_1 \, r_2} \, dS \qquad (18)$$

wherein dS is an element of surface of the ventricular muscle mass upon which one path of the kind S′ terminates, and integration is taken over the elements which include the set *f*. In these relations the quantity $1/4\pi K$ is taken as unity, K being the specific conductivity of medium. The integrand will vanish for any path in which $m = G'/A = 1$ or $r_1 = r_2$.

If in place of the local path S′ = (e . . . f) we consider the uniform path S = (A . . . B) as the axis of the average ventricular fiber with respect to the potentials $\vec{E}_x$, $\vec{E}_y$ and $\vec{E}_z$ for which $\vec{r}$ is constant, we have

$$\int_T^S \{ \vec{E}_x + \vec{E}_y + \vec{E}_z \} \, dt =$$

$$s\vec{A}_T = (n + 1) \, \vec{A}_\rho + (n - 1) \, \overrightarrow{\rho B} \qquad (19)$$

wherein n is defined by the relation

$$n = \frac{\pi}{2S} \int_B^A \nabla T_o \cdot \vec{dS} = \frac{1}{2r} [T_A - T_B] \qquad (20)$$
$$= sG/sA_{QRs}$$

Here, for geometrical convenience S is taken as a semicircular arc, Fig. 1(*b*). We thus observe that S varies over the sphere of radius $r = sA_{QRs}/2$ about O while ρ is any point on the sphere Fig. 1(*c*).

SUMMARIO IN INTERLINGUA

Le processo regressional cardiac es definite per considerar le regression tanto contra como etiam con le gradiente de un scalar T_o (i.e. le duration del stato de excitation in ulle puncto) super un uniforme via local que es sequite per le excitation ab un arbitrari puncto ρ de stimulation inter le punctos terminal e, f situate al superficie ventricular del via. Ultra su orientation, le contribution al area de T per regression super un via arbitrari es $\vec{R} = (m + 1)\,\vec{e\rho} + (m\text{-}1)\,\vec{\rho f}$, ubi $m = G'/A$, i.e. le proportion inter le magnitudes del gradiente local e del processo de accession local. Le variationes directional $-\nabla T_o$ del stato de excitation es necessarimente uniforme a transverso le via local. Illos ha un magnitude equal al differentia inter le durationes T_e e T_f de T_o al punctos terminal del via e es independente de ambe durationes del stato de excitation inter le superficies ventricular e le puncto del stimulation. Iste tractos essential es simile al tractos definite per Wilson e su associatos pro le excitation super le ficticie fibra ventricular median. Excitation super un via local, super un submultiple de illo, o super le ficticie fibra median non pote esser calculate le un ab mesurationes o considerationes super un altere.

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SPREAD OF ACTIVATION IN THE LEFT VENTRICULAR WALL OF THE DOG. IV.

Two and Three Dimensional Analysis

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INTRODUCTION

IN PREVIOUS papers[1a,2,3,4] methods were described permitting the investigation of the activation in different layers of the left ventricular wall of the dog.

It was shown that the activation patterns of the inner and outer layers differ profoundly. Results of other investigators[1b,1c] disagree in some points with our results. The experimental techniques are not entirely comparable, however.

Considered in an endo-epicardial direction the inner layers are activated within a very short time interval. This is in contrast to the outer layers of the left ventricular wall, where an activation front propagates continuously to the epicardial surface. This front is sharply bounded and propagates through approximately the outer three-fifths of the ventricular wall. The distance between the sources and sinks of the activation wave in these layers is ordinarily less than one millimeter. A hypothesis accounting for these facts was given. It was assumed that a fast conducting (approximately 2½ m./sec.) system (Purkinje system) activates the inner layers of the wall, causing the activation of these layers within a small time interval.

In the outer layers the Purkinje system is absent, activation occurs through muscle conduction, with a smaller velocity (approximately 50 cm./sec.). The same value of 50 cm./sec. was found for the conduction velocity in isolated papillary muscles of the cat.[5]

Lewis[6] has shown that the activation of the endocardial layers of the heart is caused by the Purkinje system. Recently, Draper and Weidmann[7] working with isolated fibers found a velocity of impulse conduction of 2 m./sec.

We restricted our experiments to the lateral part of the left ventricular wall, which has a more or less constant thickness. Our results make it probable that the inner layers of this part are activated in a general direction from the

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apex to the base by the Purkinje system. The difference in conduction velocity between the Purkinje system and the myocardial fibers becomes manifest in the form and angle of the activation front in the outer layers. The propagation toward the epicardium lags behind the propagation toward the base. This can be compared with the waves caused by a fast traveling boat, in which case the waves reach the border (epicardium) long after the passing of the boat.

TWO AND THREE DIMENSIONAL ANALYSIS

These assumptions resulted from the experiments with one needle electrode, in which 8-10 separate lead points were situated.[3] To analyze the two and three dimensional properties of the activation pattern, two or three needle electrodes were introduced into the free part of the left ventricular wall. It can be expected that many difficulties will arise with this experimental approach. The complicated anatomic structure at the apex, where the ventricular septum and the bases of the papillary muscles are located very closely together, makes it impossible to define exactly the position of the needles. Moreover, the activation front will have a complicated form in this region. For this reason we restricted our experiments to the relatively simple free part of the left ventricular wall, bounded at the auricular side by the atrioventricular sulcus and at the apical side by the upper part of the insertion of the papillary muscle. Great care was taken to ensure that the tip of the needle was located in the cavity and was free of the papillary muscles.

ESTIMATION OF THE ANGLE OF THE ACTIVATION FRONT IN THE OUTER LAYERS

It is possible to measure the angle of the activation front (Fig. 1) in the outer layers, by introducing two needle electrodes at a certain distance perpendicular to the wall, in a plane, that is parallel with the direction of the propagation of the activation front (Fig. 2). We can expect that the potential differences between two lead points situated at the same distance from the activation front will be small at every moment during the activation.

In previous papers[1a,4] it was shown that the activation front has a sharply defined boundary in the regularly activated layers. If such a front were a plane it could be expected that a high potential of a short duration would be recorded when the front passes two points, connected by a line, which makes a small angle with the activation front. Because of irregularities in this front this does not happen and small irregular complexes are recorded in this case. This shows that the front is not a plane in a geometrical sense; there are small bulges. It was also found that the angle of the activation front with the epicardial surface is small.

These facts account for a number of findings. In all experiments it was found that bipolar leads between one-needle lead points, situated in the regularly activated layers, give rise to regularly formed complexes with the same polarity. To avoid confusion one-needle lead points were always connected with the registering apparatus in such a manner that negativity of the innermost lead point gave rise to an upward deflection in the record. In two-needle experiments the lead points were connected in a variable manner.

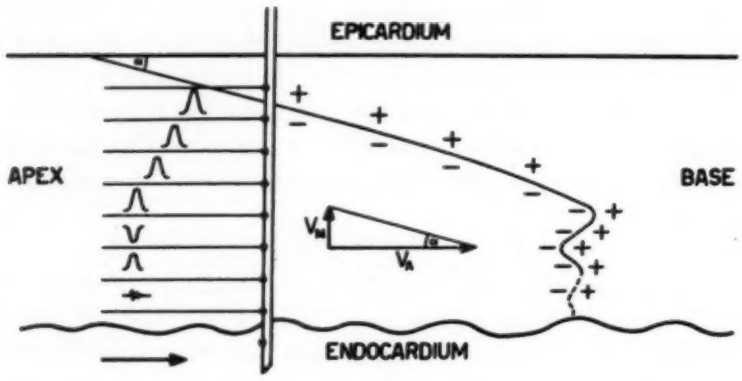


Fig. 1.—Represents a longitudinal section of the ventricular wall, with a needle electrode perpendicular to the epicardial surface. Seven of the eight lead points are lying in the ventricular wall. The complexes between the successive lead points are given at the left side of the needle. The dipole layer making the small angle with the epicardial surface is the activation front in this section, proceeding from apex to base. In the inner layers the front is nearly perpendicular to the endocardial surface. Because of the greater velocity in the Purkinje system, with respect to the myocardial conduction velocity, the activation wave forms in the outer layers a small angle with the epicardial surface. This angle follows from the construction in the figure.

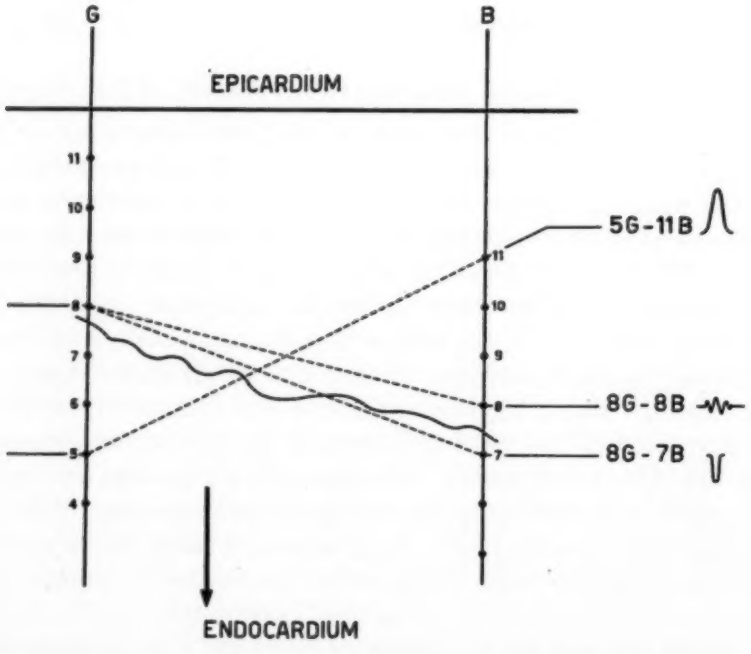


Fig. 2.—Two needle experiments (scheme). Two eleven-needle electrodes (G and B) were introduced into the left ventricular wall, perpendicular to the epicardial surface, at a distance of 2 cm. All lead points are lying in the regularly activated part of the heart wall. When the connecting line between two lead points of different needles is parallel to the activation front; only small and irregular complexes will be recorded. If the connecting line is more or less perpendicular to the front, large complexes will be seen, comparable with those between two distant lead points of one needle.

Between two lead points of different needles, complexes of either positive or negative polarity will be found. This depends on whether the one lead point or the other lead point is passed first by the activation front. If the connecting line of the two lead points is perpendicular to the activation front we can expect a regularly formed wide complex. It will be of the same polarity as a complex between the innermost of the two lead points considered and a lead point of the same needle situated close to the epicardial surface (Fig. 2). The width of these last complexes is only slightly less because of the small angle of the front with the epicardium. The potential of such a two-needle complex will not differ much from a one-needle complex. This is clearly shown in the leads 7b – 11b; 5g – 9g; 5g – 11b, of Fig. 3.

After the introduction of the needle electrodes, the lead points situated in the regularly activated region were determined. The following criteria were used.

1. The complexes between two successive lead points must have a regular form.

2. The complexes between lead points of one-needle at a greater distance must be broader in proportion to this distance.

Then we tried to find two lead points on different needles between which a small, irregular complex could be recorded. We assumed that the plane between these two lead points was parallel to the front. Between two other lead points lying in other muscle layers of that part of the wall investigated but in a parallel plane, small complexes could also be demonstrated.

In Fig. 3 curves of a typical experiment are shown. The position of the needles is the same as in Fig. 2. The two needle electrodes were called G(reen) and B(lue). From Fig. 3 it follows that lead points 7 to 11 of B and 5 to 9 of G are lying in the regularly activated layers. The two-needle leads 7g – 7b and 8g – 8b are remarkably small and irregular. 5g – 11b has already been mentioned. The polarity of the complex proves that 5g has been passed at an earlier moment than 11b. From the leads 8g – 7b and 9g – 8b it follows that the lead points of the B-needle have been passed earlier than those of the G-needle: a rather small complex of reversed polarity is shown in both leads.

In these experiments the needles were not moved from their position when the experiment was terminated. Anatomic control therefore was possible.

It follows from these experiments that the angle of the activation front with the epicardial surface is only small and approximately 5 – 10°. This is in accordance with the ratio of the conduction velocities in the Purkinje system and in the myocardium.

It has been shown in our second paper[3] that the activation pattern at the epicardial surface is very complicated: in the lateral part of the left ventricular wall the general direction of propagation of the activation is from apex to base. In small regions of this area this is not the case, however, and the front may propagate in all directions. This is easily accounted for by the small angle between the activation front and the epicardial surface and the very plausible assumption that there are small bulges in the front. In addition the wall is

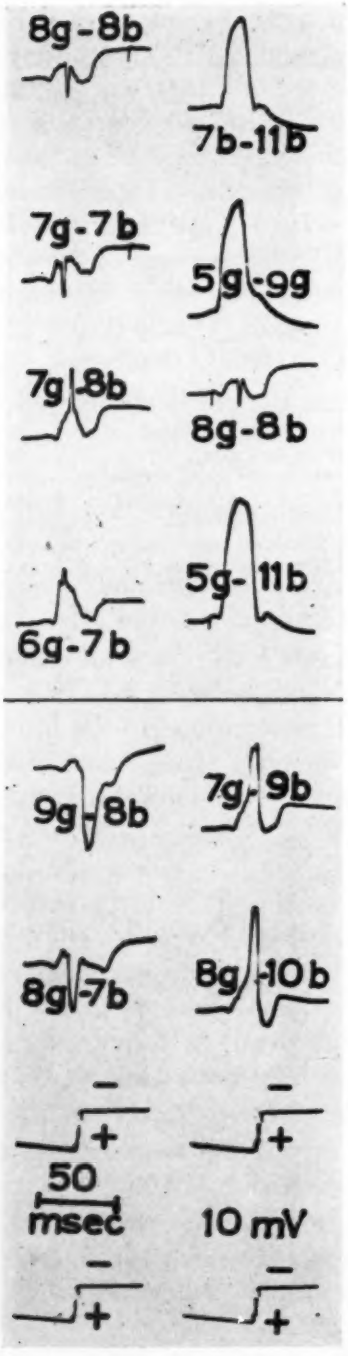


Fig. 3.—Typical example of a two-needle experiment (See Fig. 2). In this figure, as in all other figures, the negative sign at the upper side of the base line means that for an upward deflection, the electrode or lead point at the left of the indicated lead points is negative with respect to the electrode or lead point at the right, e.g., in 7b-11b, 7b is negative with respect to 11b; in lead 8g-8b, 8g is positive with respect to 8b. Of course these actually recorded complexes are not as regular and constant as in the scheme; the whole picture, however, agrees very well with our assumptions. A closer analysis is given in the text.

not equally thick everywhere, and the Purkinje system probably does not penetrate to the same depth everywhere. At the surface a small bulge in the front will arrive first. From this point the activation wave will propagate over short distances, until regions are reached that had been activated by waves coming from another bulge, having reached the epicardial surface at another point. Therefore, measuring propagation velocity at the epicardial surface gives rise to great inaccuracies even when the lead points are at a greater distance apart.

GEOMETRICAL PROPERTIES OF THE ACTIVATION FRONT IN THE INNER LAYERS

As has been shown, the fixation of the needle electrode was sufficient to allow a study of the geometrical properties of the front with two needles. The fixation was even good enough to permit experiments of long duration (Fig. 5 of Ref.[4]). Therefore, it was decided to perform experiments with three needles.

We are well aware that because of the very complicated anatomic properties at the cavital side of the ventricular wall, i.e., the trabeculae carnae, etc., and the unknown intramural extension of the Purkinje system, the interpretation of the results of the three-needle experiment must be considered hypothetical. We propose to give a working hypothesis as a stimulus to further investigation and also to discussion.

The fact that the activation in the inner layers in endo-epicardial direction is practically simultaneous suggests that the Purkinje network is dense or that the myocardium has a greater conduction velocity in this region. This last assumption is very improbable as will follow from our results with extrasystoles, to be mentioned.

The Purkinje system activates the portions of the ventricular muscle lying within this network.[4,8] This occurs most probably in all directions from the transition between the Purkinje fibers and the myocardial fibers. The activation wave in the ventricular muscle originates from this point and spreads in all directions. Therefore, activation fronts will be present propagating in a direction contrary to the general direction of the Purkinje system in that part of the wall considered. These fronts will certainly collide with activation processes that have originated at an earlier moment. Between lead points lying at short distances in the inner layers no well-developed complexes can be expected, since no well-organized activation front will be present between these points. Although the velocity in the Purkinje system is great, there will still be time differences when larger regions are considered. Therefore, an activation front in the small muscle regions propagating in the general (apico-basal) direction will have less chance to meet an already depolarized region than one going back. An excess of activation fronts with apico-basal direction can be expected, showing itself in well-developed complexes between needles placed at a greater distance. The potential of these complexes is determined by the statistical geometrical properties.

The intramural extension of the Purkinje system makes it probable that the angle of the front in the inner layers with the endocardial surface will be approximately 90°.

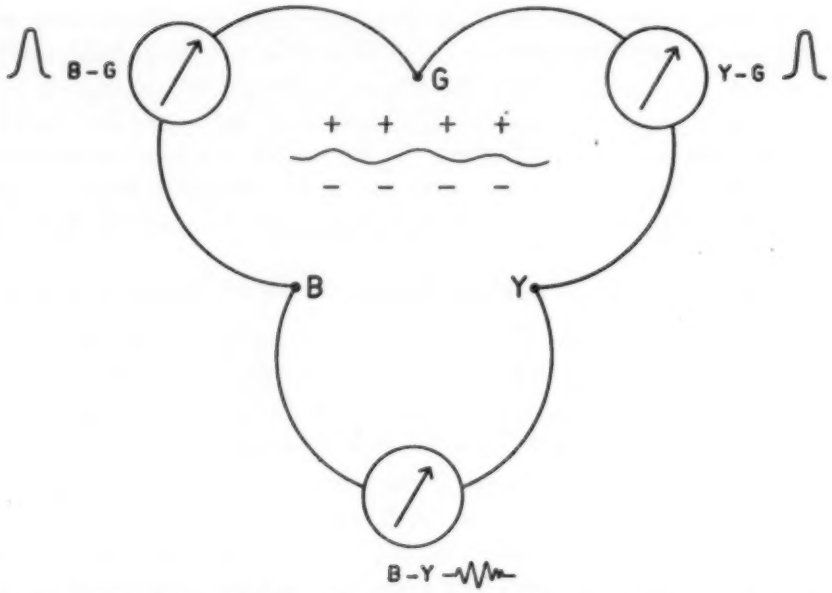


Fig. 4*a*.—Three-needle experiment. The needles are indicated as B(lue) and Y(ellow), lying nearer to the apex than the other needle G(reen), which is closer to the base. The connecting line of B and Y is parallel to the atrioventricular sulcus. A part of the activation front, spreading from apex to base (from B and Y to G) in this part of the wall is indicated as a dipole layer.

The complexes between lead points in the inner layers, which can be expected when the front in these layers is propagated from apex to base, are given.

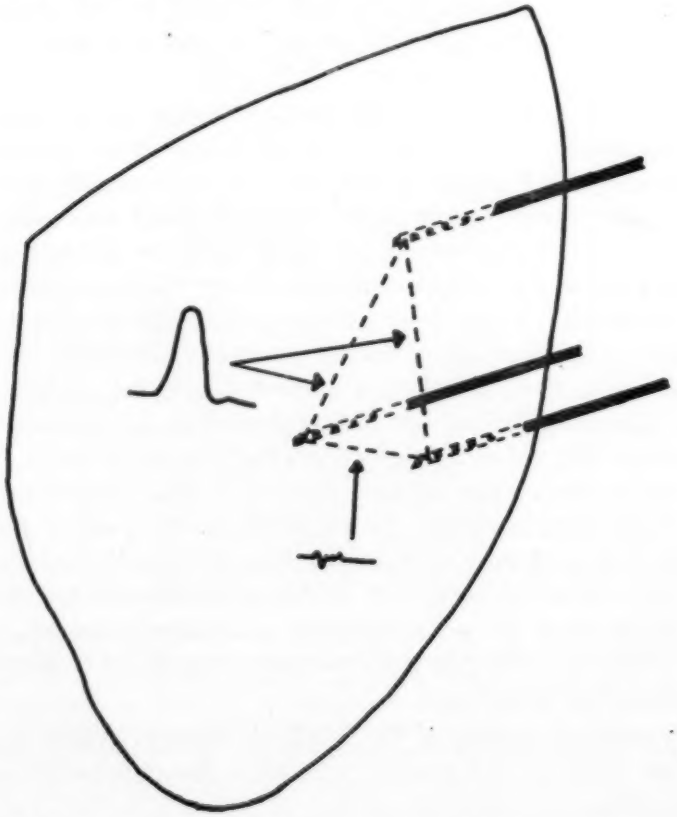


Fig. 4*b*.—Same as Fig. 4*a*, but now three dimensions are given.

Three needle electrodes were inserted, schematically shown in Fig. 4, *a* and *b*. The two needles B(lue) and Y(ellow) were introduced at the lateral side of the left ventricle nearly one-half way between apex and base. A third needle was introduced 0.5 cm. from the atrioventricular groove, thus forming an equilateral triangle with the first mentioned two needles. With great care the lead points in the inner layers of the ventricular wall were located. The bipolar complexes of each needle are shown in Fig. 5. They are relatively small and irregular. The two-needle complexes are given in Figs. 6 and 7. The difference in form of the complexes of the B and Y needles, which are small and irregular, and those of the B and G and of the Y and G needle electrodes, which are large and well developed, are obvious.

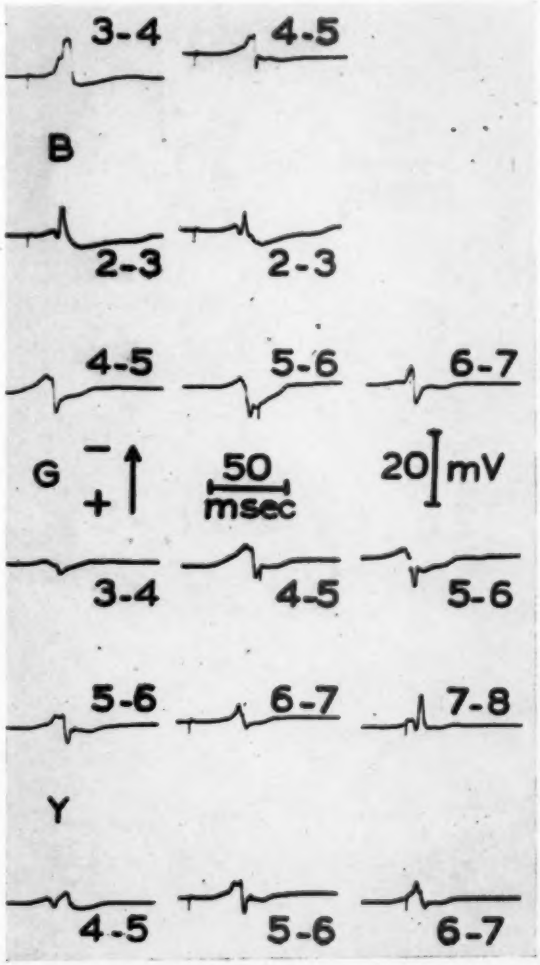


Fig. 5.—The bipolar complexes of the B, G, and Y needle electrodes show that with great probability lead points 2 and 3 of the B needle electrode; 3, 4, and 5 of the G; and 4, 5, and 6 of the Y needle electrode are lying in the inner layers.

The genesis of these complexes is shown schematically in Fig. 4*a*. It is noteworthy that many complexes between different lead points of the B and Y needle electrodes show nearly the same shape. Leads 5B – 6Y and 5B – 4Y give a somewhat larger, less bizarre complex. (Note that the lower complexes

of both rows have been recorded with a higher sensitivity.) It seems probable that the position of lead point 5B is outside the inner layers. It may be concluded from this experiment, (representative of a series of three) that for the inner layers longitudinal conduction is most probable and that the front is perpendicular to the general direction of propagation. It is emphasized that these results are only valid for this free part of the left ventricular wall. Fig. 8 shows a complex at the beginning of which a Purkinje spike probably occurs. (The lower of the two rows has been recorded with a higher sensitivity.)

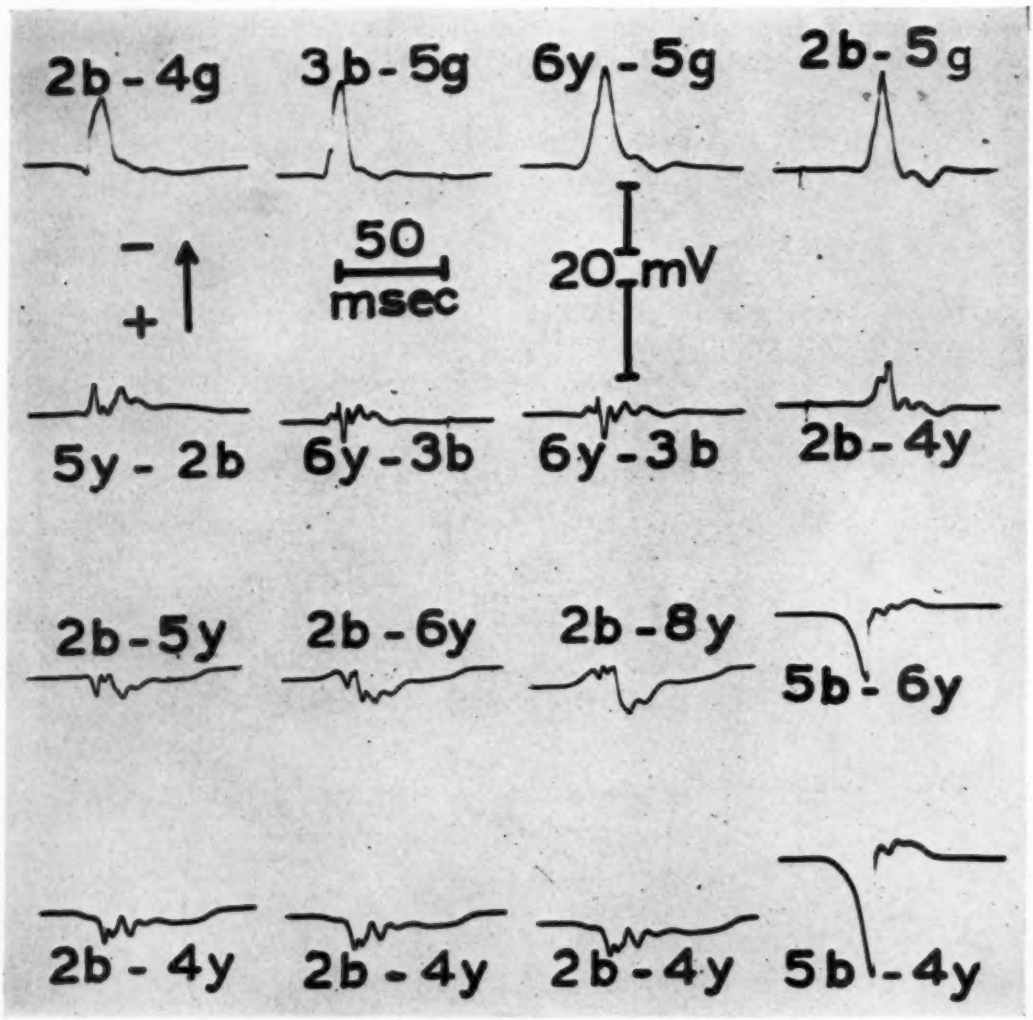


Fig. 6.—Complexes between different needle electrodes. The complexes between the *B* and *Y* needle electrodes are small and have an irregular form. They are only slightly dependent on the choice of the lead points, suggesting a front parallel to the needles. The b–g and y–g complexes, however, are well developed. Complex 5b–4y is already larger, which may be caused by the fact that 5b is already in the outer layers.

EXTRASYSTOLES

To check the hypothesis mentioned in this and in previous papers, extrasystoles were elicited by electrical stimulation. To be sure about the

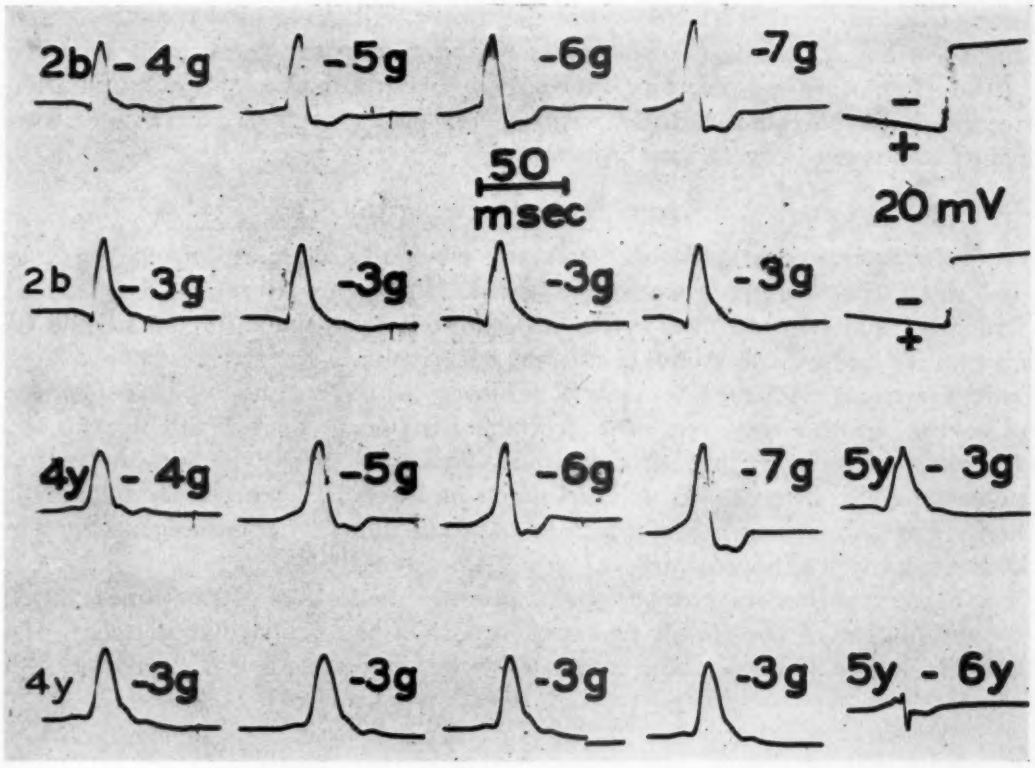


Fig. 7.—Here the complexes between lead points situated in the inner layers but parallel to the direction of propagation of the front are given. Large, well-developed complexes are seen.

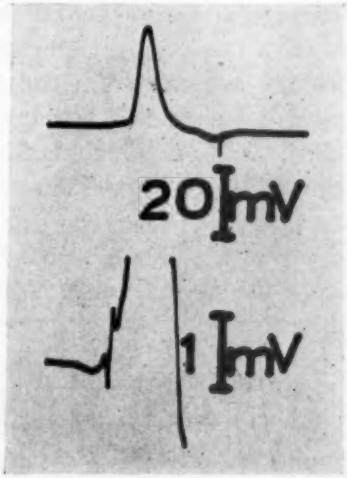


Fig. 8.—Complexes between two lead points of different needles. The lead points are situated in the inner layers. The small spike at the beginning of the complex is probably a Purkinje spike. The two leads are identical, only the sensitivity of the lower curve is increased, twenty-fold.

geometrical properties, lead points of the same needle were used for stimulation and recording. In this manner we could be sure about the spatial relations of the stimulating and recording lead points. To locate the point of stimulation accurately, anode and cathode were placed close together. Successive lead points of one needle were mostly used.

TECHNIQUE OF STIMULATION

Stimulator.—A stimulator developed especially for physiologic purposes and simulating a current source was used. Frequency, duration, and current strength could be controlled within wide limits. Also single impulses could be given. To reduce the stimulus artifact effectively, a transformer with a high inductance and a 1:1 ratio was used. This caused the output to be independent of earth. In this way overloading of the amplifier, which would disturb the recording for a long time after each stimulus, was prevented and the action potential could be recorded without much interference from the stimulus artifact. For single impulses a duration of approximately 2 milliseconds and a current strength of approximately 100 μ A were mostly used.

These experiments were performed to study the form of extrasystoles caused by stimulation of the epi- or endocardial part of the ventricular muscle. The possible implications of these findings for our hypotheses will be considered.

ENDOCARDIAL STIMULATION

With stimulation at the endocardial side two possibilities are obviously present.

1. The stimulus excites the Purkinje system and throughout the whole wall the form of the bipolar leads will not differ much from the complexes of a normal beat; the reversal phenomenon[4] could still be present.
2. The Purkinje system is not activated but only the muscle fibers.

In that case the small irregular complexes of the inner layers will change into larger ones comparable with the normal complexes in the outer layers and the front passes with a constant velocity from the point of stimulation in the inner layers toward the epicardial surface. It can then be expected that the reversal phenomenon, probably due to activation of a certain layer in epi- endocardial direction, will disappear. The ventricular wall as a whole will show a uniform activation pattern.

In these two cases the complexes in the outer layers will not differ much, either from each other or from the normal ones, because a sharply defined activation front is present in all cases.

EPICARDIAL STIMULATION

Since there is no Purkinje system in the outer layers, the epicardial extrasystoles always start with muscle activation. If this caused activation of the Purkinje system in the inner layers, the complexes of intramural bipolar leads would have an irregular shape, as with normal beats. If this did not occur, the whole wall would show the same picture. These epicardial extrasystoles would then give rise to complexes in the inner layers of the same shape and polarity as in the outer layers.

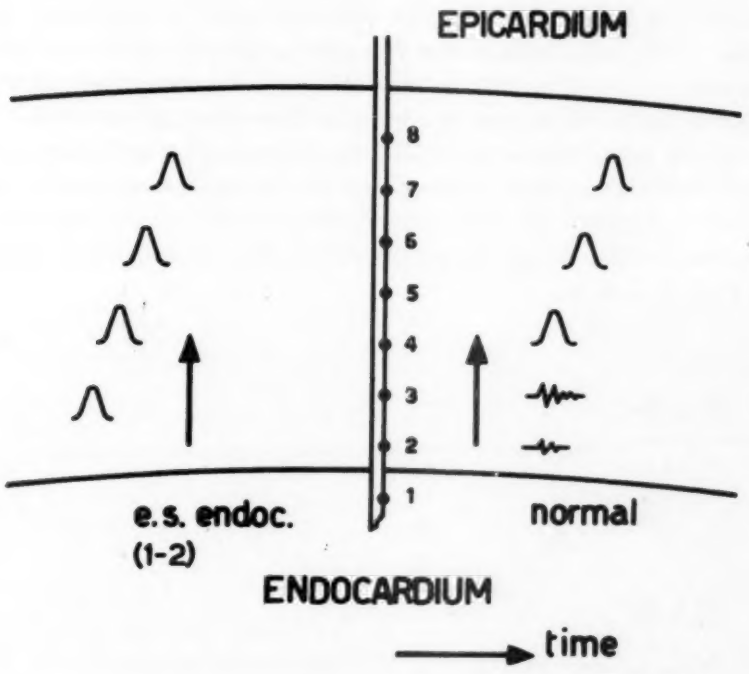


Fig. 9.—Schematic representation of the form of intramural bipolar complexes during stimulation of endocardial layers.

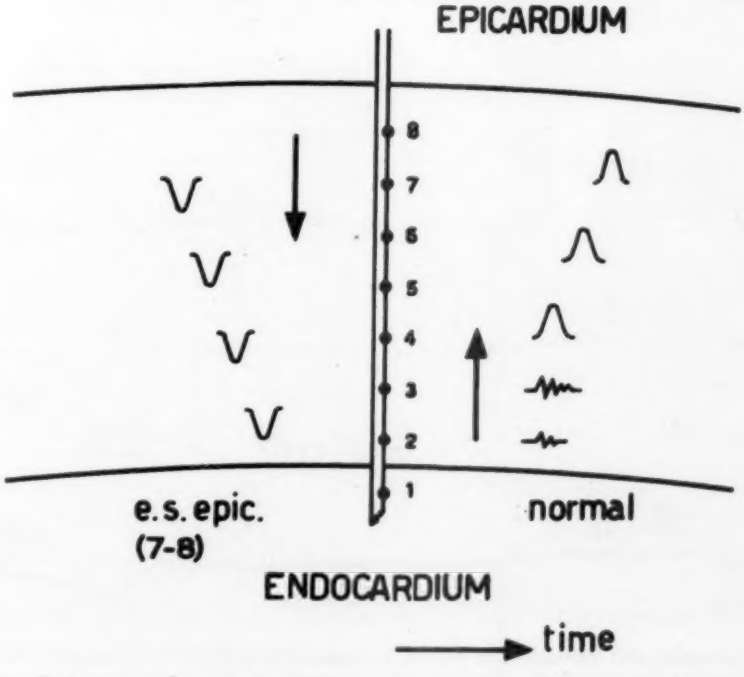


Fig. 10.—Schematic representation of the form of intramural bipolar complexes during stimulation of epicardial layers.

All complexes would change their polarity, with exception of the reversal phenomenon. The layer where this happens is already activated in epi- endocardial direction during normal beats. If muscle conduction alone occurred, equal distances would be passed in an equal time everywhere in the wall. The complexes would have the same width throughout the wall, and in the outer layers they would show a mirror image of the complexes of normal beats, when the propagation velocity in both directions was the same. The figures show a number of recordings from several experiments. A schematic representation is given in Figs. 9 and 10.

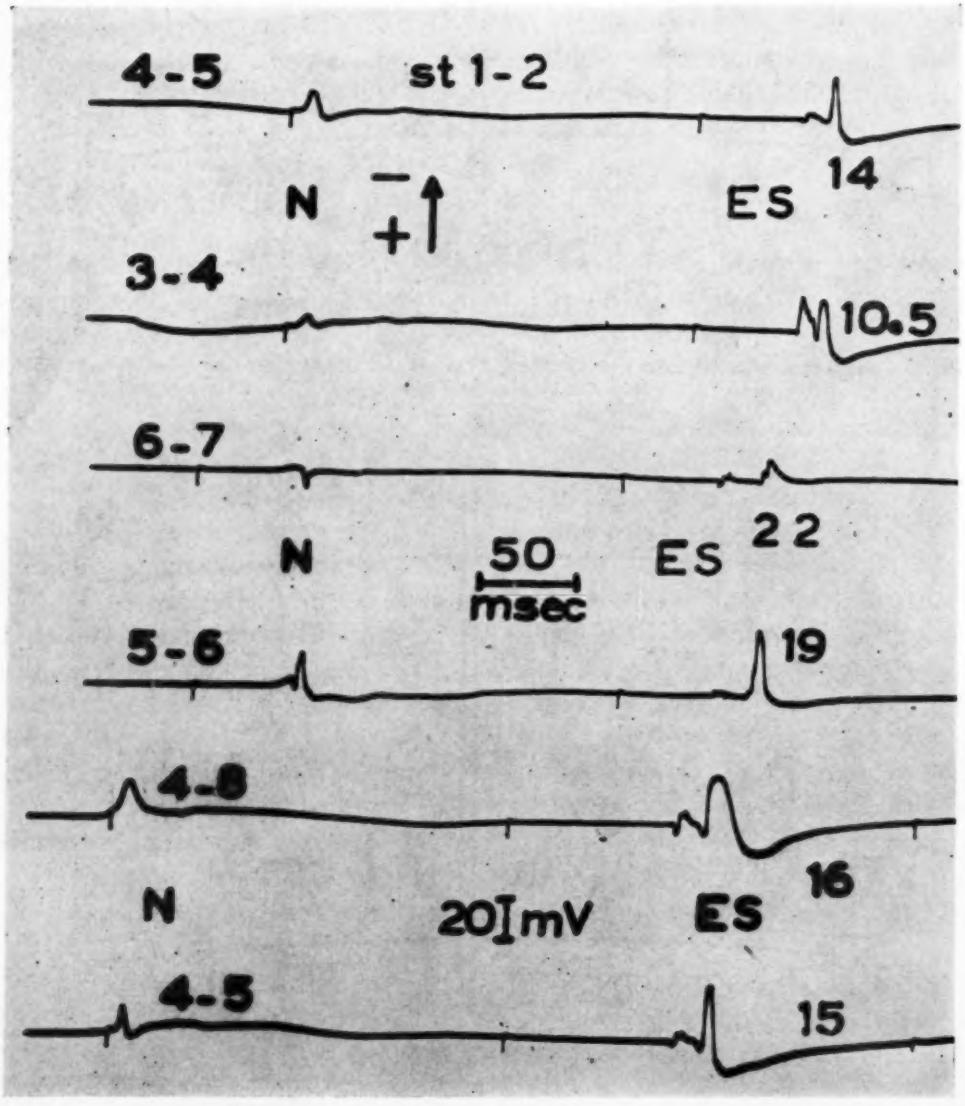


Fig. 11.—Endocardial extrasystoles evoked by stimulation of the inner layers. Lead points 3, 4, and 5 are lying in the inner layers. The bipolar complexes of the normal beats occur almost synchronously. The numerals near the extrasystolic complexes give the delay in milliseconds; this applies also to the following figures. The delay is given as the time between the stimulus artifact and the starting of the complex. In some cases a variation of some milliseconds may occur as the stimulus is given at an arbitrary time during each cycle. The bipolar complexes of the extrasystoles occur successively, presumably by muscle conduction. Note 4–8 which is not much higher but has a greater width.

RESULTS

With *endocardial* stimulation we did not succeed in recording complexes in the inner layers caused by the excitation of the Purkinje system, but a few times they could be seen on the fluoroscopic screen. The reversal phenomenon always disappeared with these endocardial extrasystoles.

In Fig. 11 the normal complexes of the inner layers are small and irregular, and there is no significant time difference. The extrasystoles, however, have well-developed complexes starting 3½ msec. later for each lead point. The

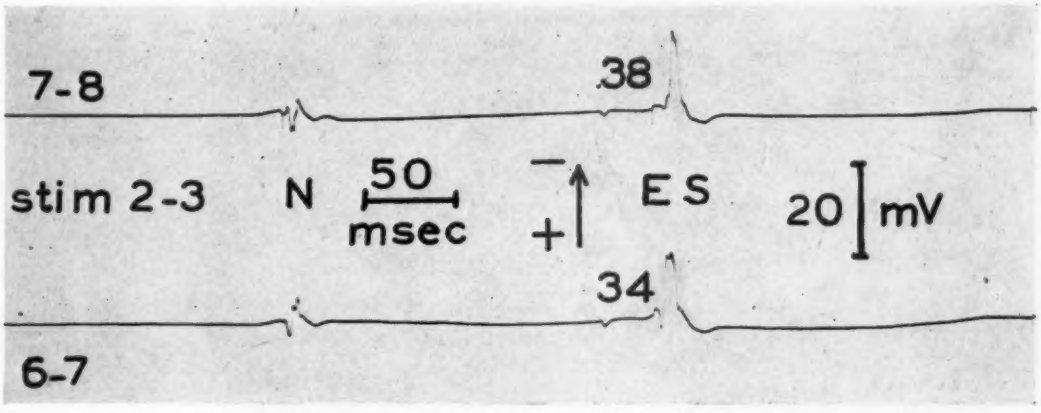


Fig. 12.—Endocardial extrasystoles. Lead points 6, 7, and 8 are situated in the inner layers. Very irregularly formed synchronous complexes change into larger successive ones and have a polarity which can be expected when the inner layers are activated by muscle conduction in endo-epicardial direction.

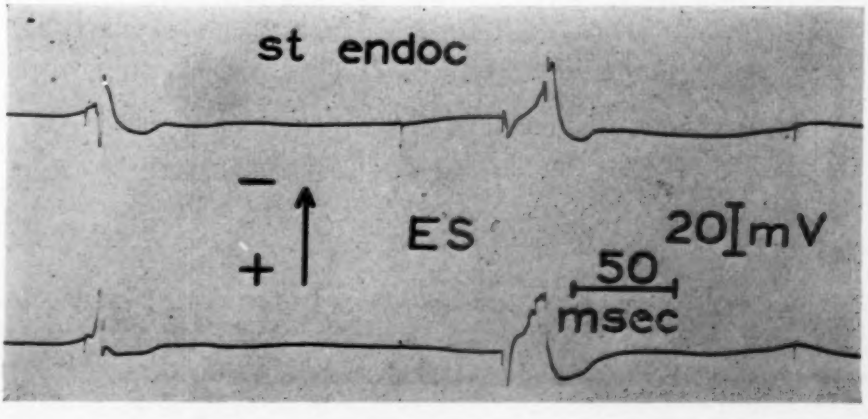


Fig. 13.—Stimulation of endocardial layers. Lead points situated in these layers. The upper complex of the normal beats has a reversed polarity with respect to the complexes of the consecutive epicardial layers. During extrasystoles the polarity reverses, pointing to endo-epicardial activation of the layer in which the lead points are situated. The extrasystolic complex has an irregular form.

distances between successive lead points are 2 mm. The propagation velocity in endo- epicardial direction is therefore 60 cm./sec. The complex 4 – 8 of the third series is definitely longer (approximately 20 msec.) in relation to the greater distance between the lead points. The complex 6 – 7 is not well developed, for unknown reasons. Fig. 12 shows very demonstratively the transformation of a very irregular normal complex into a regular tracing during an extrasystole.

Fig. 13 shows a case in which the reversal phenomenon disappears during endocardial stimulation. The extrasystoles for both leads, formed rather irregularly, are very much alike, while the normal beats here give complexes of an opposite polarity.

Epicardial stimulation gives rise to muscle conduction only, both in the recordings and on visual observation. The Purkinje system in the inner layers of that part of the wall considered is not activated.

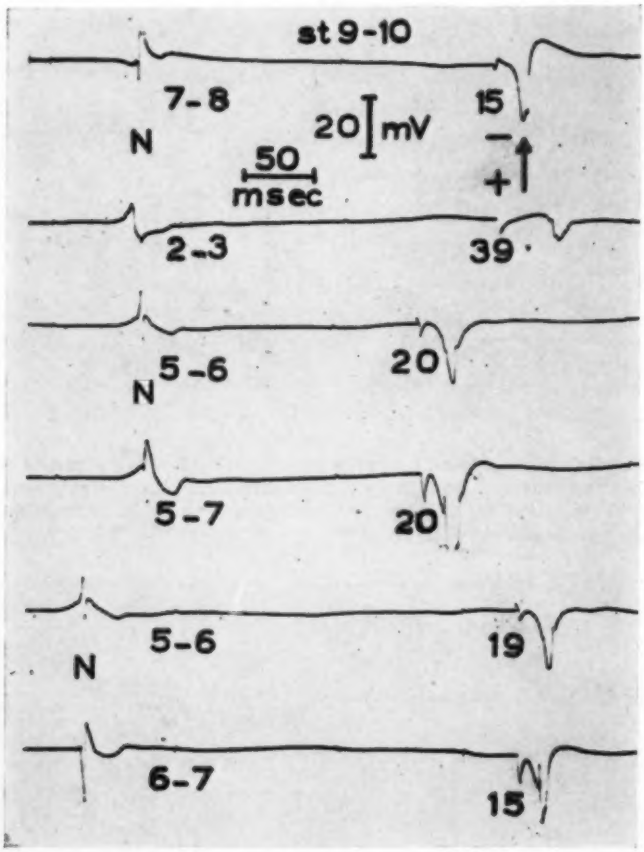


Fig. 14.—Epicardial extrasystoles. Stimulation between lead points 9 and 10. The slightly irregular complexes between the consecutive lead points change into complexes with reversed polarity, pointing to activation in epi-endocardial direction, except the complex between 6 and 7 where during normal and extrasystolic beats reversed polarity remains (reversal phenomenon).

The first row of Fig. 14 demonstrates that sometimes there is a small latent period of some milliseconds after the stimulus, before the muscle fibers are excited and conduction occurs, for the distance between 7 – 8 and 9 – 10 (the stimulating electrodes) does not account for the long delay of 15 msec. If we extrapolate the delays up to the stimulating electrodes we find a latency time of approximately 4 msec.

In other experiments on the isolated papillary muscles of the cat sometimes a latent period of 3½ msec. has been demonstrated (not yet published).

Epicardial stimulation results in a uniform course of the activation process in all layers of the part of the wall investigated (Fig. 14). Several complexes are shown with the delays after the start of the stimulus. All complexes are alike, 2 – 3 excepted for an unknown reason. The extrasystolic complexes occur

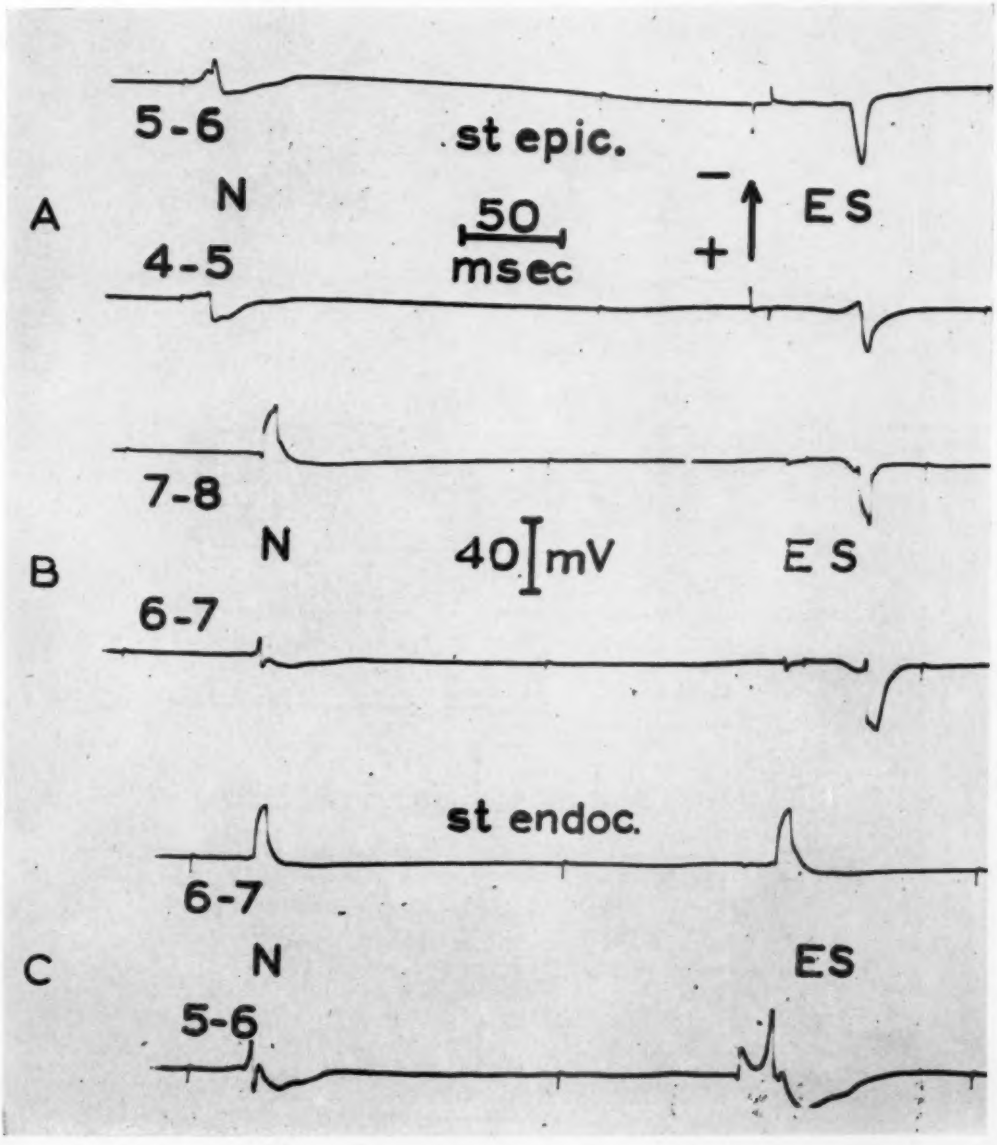


Fig. 15.—Epicardial and endocardial extrasystoles in inner layers. 6–7 seems to be displaced into the more regularly activated layers in *C*.

in consecutive layers in uniform succession. In this experiment the propagation velocity in epi-endocardial direction is 35 cm./sec. Lead 6 – 7 is a good example of a reversal complex which does not change its polarity with epicardial extrasystoles.

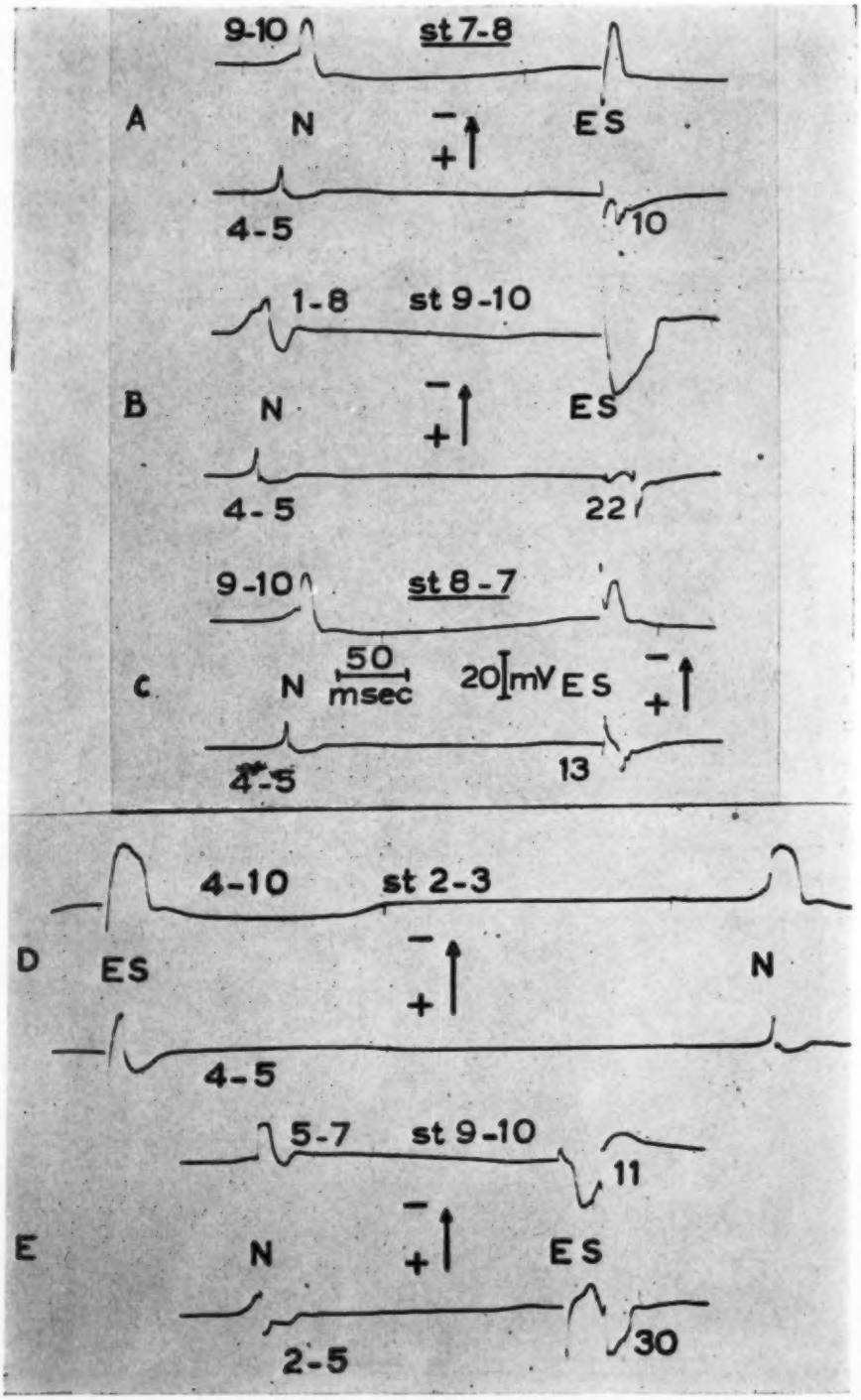
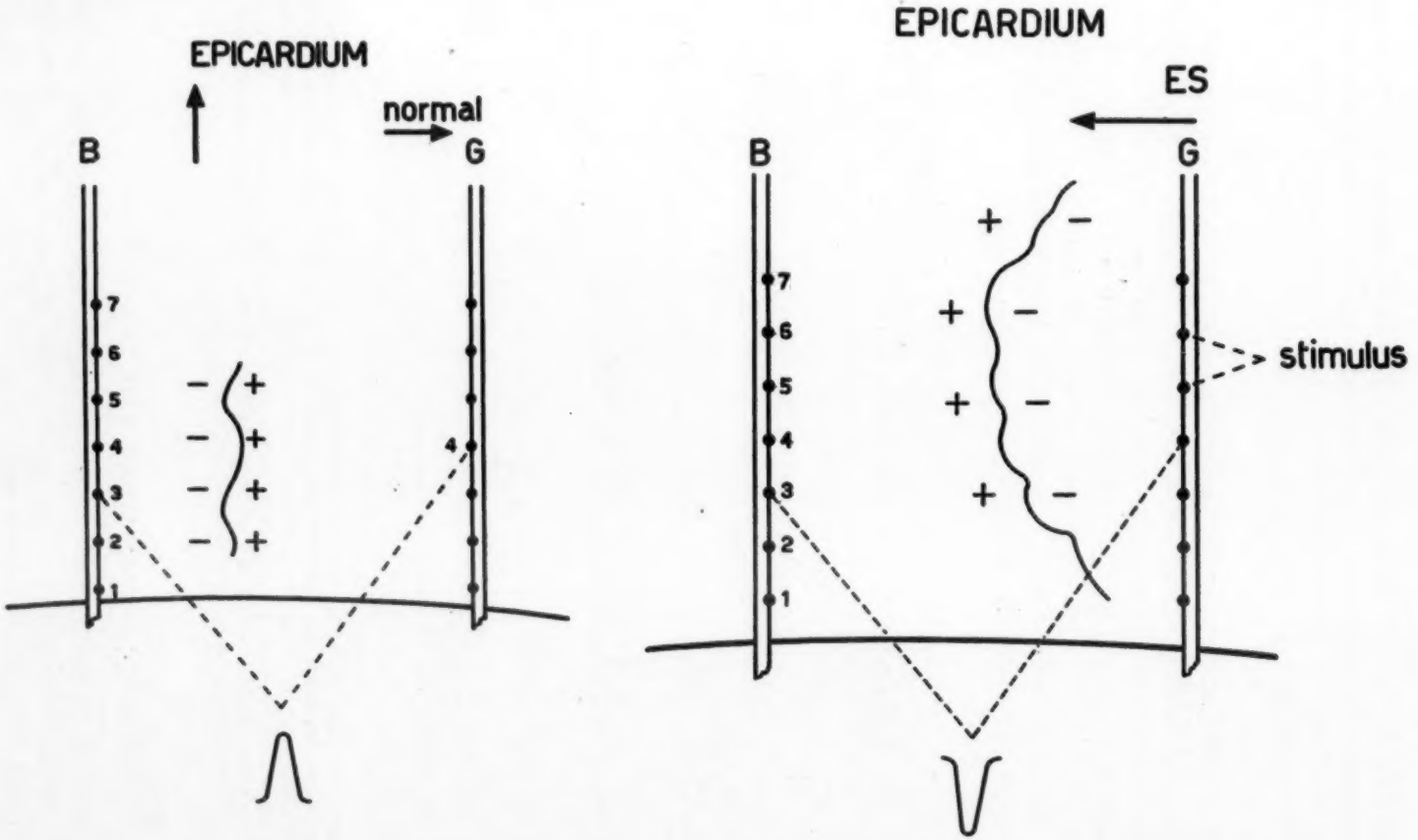


Fig. 16.—Stimulation between different lead points of one needle. In *A* stimulation occurred at electrodes situated between the recording lead points. As can be expected, in 9–10, situated in the outer layers and closer to the epicardium than the stimulating electrode 7–8, the complex has the same form and polarity as the normal beats. 4–5 has an opposite polarity. In the rows *B*, *C*, *D*, and *E*, the form of the complexes and the time relations occur as would be expected. In some cases the delay is not given; here the starting of the complex falls within the stimulus artifact.



a. *b.*

Fig. 17.—*a* and *b*. Schematic representation of two-needle experiments during which the inner layers were stimulated. In *a* the normal conditions are given. During normal beats there is tangential conduction in the inner layers. The front propagates from *B* to *G*, giving rise to a complex as indicated at the bottom of *a*. The activation conditions during stimulation through lead points of needle electrode *G* are given. The two-needle complex shows reversal of polarity (*b*).

RECORDINGS WITH STIMULATION AT VARIOUS POINTS

The upper row of Fig. 15 shows the reversal of the complexes in the inner layers with epicardial sitmulation. In the lower row the needle seems to be displaced, and 6 – 7 is now situated in the region of normal muscle conduction. The endocardial extrasystolic complex is very much the same as during the normal beat.

Fig. 16 is interesting because of the fact that stimulation was performed at different points, and leads were also taken over greater distances within the wall. Reversal of the polarity of the stimulus does not change the form of the extrasystolic complexes, as can be seen in the first and third rows. Complex 2 – 5 shows with normal beats a poorly defined picture; the epicardial extrasystole changes this into a well-developed complex. The velocity of propagation is in agreement with the already mentioned values.

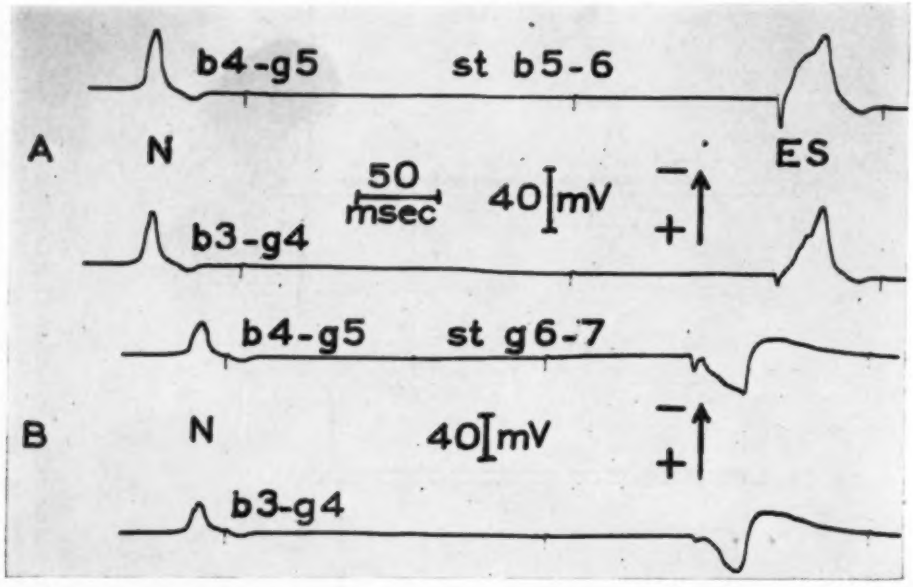


Fig. 18.—Complexes during a typical two-needle experiment in the inner layers. The extrasystoles have the expected form. On stimulation of the needle electrode that is passed last in a normal beat, the complexes reverse their polarity. Stimulation through lead points of the needle electrode passed first gives rise to complexes with the same polarity as in normal beats. The extrasystoles are broader; the reason is that the slow-conducting muscle system is activated near the stimulating electrode, not the Purkinje system. This system is probably activated at a later moment.

ANALYSIS OF EXTRASYSTOLES WITH TWO NEEDLES

Further progress in the analysis of the propagation of extrasystoles in two and three dimensions was achieved by the insertion of two needles. The lead points in the inner layers were determined.

In Fig. 17,*a* and *b*, the schematic complexes are shown that can be expected between two endocardial lead points of two different needles (B and G). The G-needle normally is reached later by the Purkinje activation wave than the

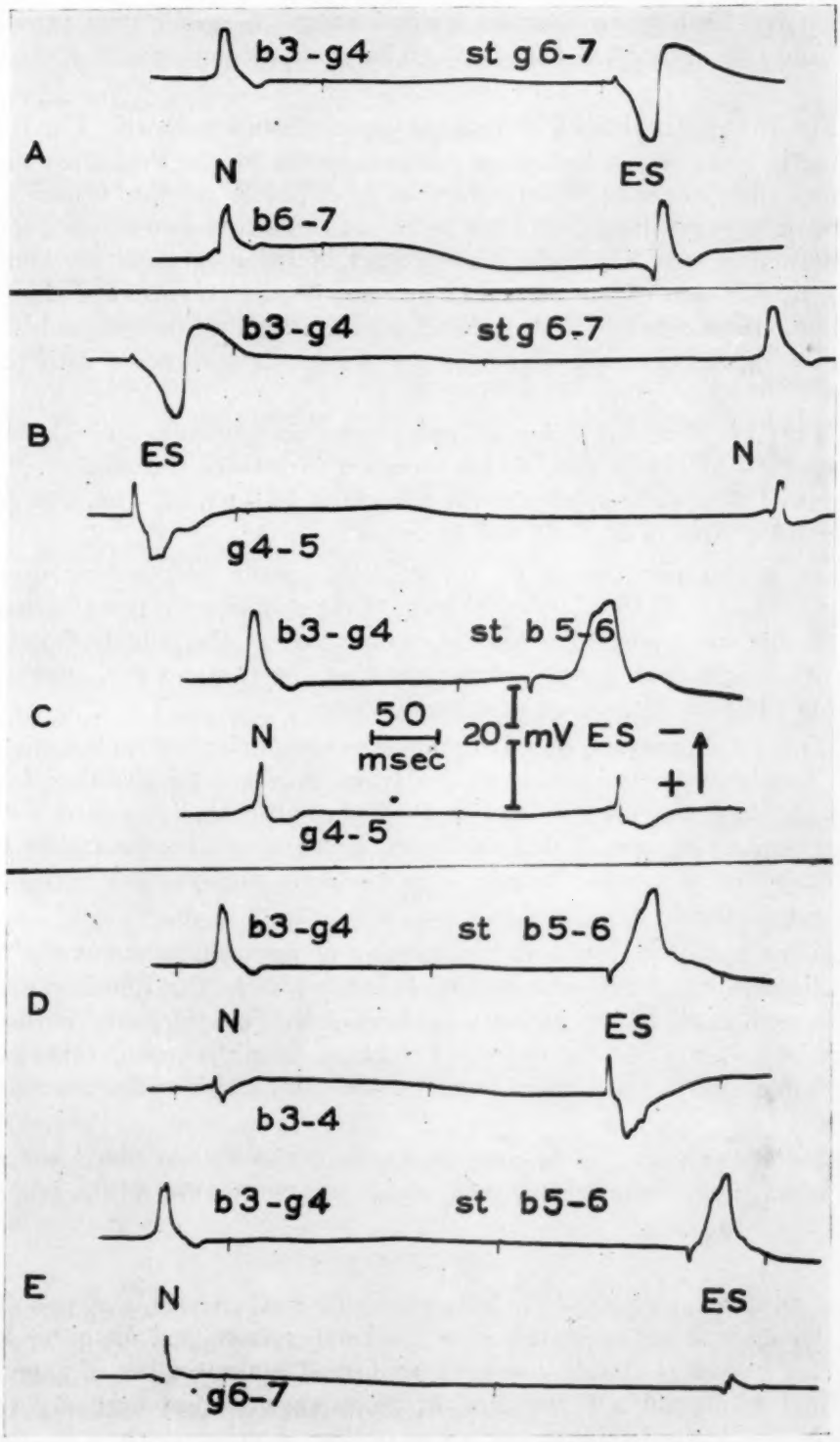


Fig. 19.—More examples of two-needle extrasystoles are given. The B-needle is passed first by the activation wave in the Purkinje system, the G-needle is passed later on. Stimulation of lead points on the B-needle (*C*, *D*, and *E*) gives rise to bi-needle complexes with the same polarity as during normal beats. Bipolar complexes (between lead points on the nonstimulating needle electrode, *C*) do not differ much from the normal complexes. At this distance from the stimulus the activation wave is most probably conducted by the Purkinje system. In *E*, however, there are differences.

B-needle (Fig. 17*a*). Extrasystoles elicited at the G-needle now pass in the opposite direction toward B (Fig. 17*b*) giving rise to complexes of reversed polarity.

In Fig. 18 the complexes found during experiments are shown. The B-needle is situated in a region reached at an earlier moment by the Purkinje activation wave than the G-needle. Stimulation of lead points on the B-needle gives rise to two-needle complexes with the same polarity as the two-needle complexes of the normal beats. Since the trailing part of these extrasystolic complexes is identical with that of the normal complexes, it may be supposed that at the end of the extrasystole in the considered region, the Purkinje system is excited. Stimulation of the G-needle gives rise to two-needle complexes with reversed polarity.

In Fig. 19 more examples of two-needle extrasystoles are given. The complexes b6 – b7 (lower row, *A*) are recorded with twice the sensitivity as the upper curve. The same applies to the complexes g4 – g5, b3 – b4, and g6 – g7, all in the lower rows of *B*, *C*, *D*, and *E*.

When stimulation results in an activation wave propagating in normal direction (Figs. 18, *A*; 19, *C*,*D*,*E*) the form of the complexes is pointing to propagation of this wave partly by muscle conduction (in the neighborhood of the stimulating electrodes), giving a broadening of the complex and partly to excitation of the Purkinje system at some distance.

In Fig. 19, *C* complex g4 - 5 during an extrasystolic beat is but slightly altered. The lead points considered are lying at a greater distance from the stimulating electrodes b5 - 6. In Fig. 19, *D*, however, lead points b3 – 4, which are lying close to the stimulating electrodes, give rise to a complex, that is much larger than during normal beats. This strongly suggests the occurrence of muscle conduction.

When stimulation results in propagation of an activation wave in the opposite direction compared with normal beats, probably the Purkinje system is not activated at all in the region considered. In Fig. 19, *A* the extrasystolic complex b6 – 7, recorded at a greater distance from the stimulating electrodes g6 – 7, is larger and has a more regular form comparable with a normal muscle complex.

The pictures vary for different beats, since the extrasystoles will not fall at the same time during each cycle, which causes other conditions of propagation.

CONCLUSIONS

The anatomic picture of the left ventricular wall consisting of two different layers, an inner layer activated by a Purkinje system and an outer layer in which this system is absent, has been confirmed by a number of experiments. Recordings of normal and extrasystolic beats showed that normally the activation in the inner layers occurs more or less synchronously. In the outer layers, however, a well-developed front, making a small angle with the epicardial surface, propagates uniformly with a velocity of approximately 50 cm./sec. In some parts of the wall the direction of propagation is in an apico-basal sense.

In all cases it was possible to make extrasystoles and predict the sign of the resulting complexes based on the previously mentioned picture of the left ventricular wall.

SUMMARY

Needle electrodes were used for the spatial analysis of the activation of the left ventricular wall of the dog. Two and three electrodes of this type were introduced perpendicular to the epicardial surface into the lateral part of the left ventricular wall.

With two-needle electrodes the angle of the activation front with the epicardial surface in the outer layers of the lateral, left ventricular wall, propagating in an apico-basal direction was estimated and found to be small, approximately 5 to 10°. This and the fact that the activation front has small bulges explains the complicated activation pattern at the epicardial surface.

The activation pattern of the inner layers of the lateral part was investigated with three needles. The inner layers are activated in a very short time interval, considered in an endo-epicardial direction. In an apico-basal direction the experimental findings show well-developed complexes, pointing to an excess of apico-basal propagation in small areas activated by single Purkinje fibers. This explanation must be considered as a working hypothesis.

The lead points of one needle electrode introduced perpendicularly to the epicardial surface were also used as stimulating electrodes. In this way it was proved that stimulation in the endocardial layers gives rise to an activation wave in endo-epicardial direction of all layers of that part of the wall investigated. The bipolar complexes of a needle electrode had approximately the same form and duration in all layers, indicating a nonfunctioning Purkinje system in the inner layers of the investigated part.

Epicardial stimulation showed reversed complexes in all intramural bipolar leads of the investigated area.

In experiments with two-needle electrodes placed in an apico-basal direction it was shown that the two-needle complexes in the inner layers did not reverse their polarity if two of the lead points of the apical needle electrode were used as a stimulating electrode, but reversed their polarity if the basal needle electrode was used.

It was made plausible that the Purkinje system in the neighborhood of the stimulating electrode was not activated but that this occurred at a distance from the needle.

SUMMARIO IN INTERLINGUA

Electrodos del typo agulia esseva usate pro le analyse spatial del activation del pariete sinistroventricular in canes. Le electrodos esseva usate in pares o combinationes de tres. Illos esseva introducite in le parte lateral del pariete sinistroventricular, perpendicular al superficie epicardial.

Per medio de un serie de experimentos il esseva confirmate que le pariete sinistroventricular consiste anatomicamente de duo differente stratos. Le strato interior es activate per un systema de Purkinje. In le strato exterior iste systema es absente.

Registrationes de pulsos normal e extrasystolic monstrava que in le stratos interior le activation occurre normalmente plus o minus synchronemente. Del altere latere, in le stratos exterior un ben-disveloppate fronte, que forma un acute angulo con le superficie epicardial, se propaga uniformemente con un velocitate de circa 50 cm per secunda. In altere partes del pariete le direction propagational es apico-basal.

In omne casos il esseva possibile producer extrasystoles e predicer le signo del resultante complexos super le base del supra-mentionate structura del pariete sinistroventricular.

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THE LEAD VECTORS OF MULTIPLE DIPOLES LOCATED ON AN ELECTRICALLY HOMOGENEOUS CIRCULAR LAMINA

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IN THE vector analysis of the electric field of the heart, the assumption is usually made that the total electromotive force generated by the heart may be represented as arising from a single equivalent dipole. It is well recognized that this assumption has definite limitations but the degree to which it introduces error in the case of leads recorded from the body surface is controversial. A more realistic approach, and one which obviates differences of opinion, is the representation of the heart as a cluster of individual dipoles, each generating its own electromotive force.[1]

In the case of the single equivalent dipole assumption, the difference in potential, P – P′, recorded by an electrocardiographic lead, equals the scalar product of (1) the total electromotive force, expressed by the vector, $\vec{E}$, and (2) the electrocardiographic lead, possessing magnitude and direction, and, therefore, capable of being expressed as a vector quantity, $\vec{L}$. When the heart is represented as consisting of a cluster of dipoles, no assumptions whatever are made if the following equation is written for the recorded difference in potential:

$$P - P' = e_1 \vec{l}_1 + \vec{e}_2 \vec{l}_2 + \ldots \vec{e}_n l_n \qquad (1)$$

where the e terms represent the electromotive forces developed by each of the individual dipoles, and the l terms represent the different magnitudes and directions of the electrocardiographic lead with respect to each of the individual dipoles in question. This equation is justified from consideration of Helmholtz's *Principle of Superposition*, discussed elsewhere.[1-3] Although each of the $\vec{l}$ terms might remain constant during the recording of the lead, each is unknown. It is, therefore, obvious from Equation (1) that fluctuation of the difference in potential, P – P′, actually provides no direct information concerning variations in the dipoles considered either individually or totally. There are two possible assumptions which would permit simplification of this equation.

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1. If all of the individual dipoles of the cluster had the same magnitude and orientation, it is evident that

$$\vec{e_1} = \vec{e_2} = \ldots = \vec{e_n} = \frac{\vec{E}}{n}.$$

Then

$$P - P' = \frac{E}{n} (\vec{l_1} + \vec{l_2} + \ldots \vec{l_n}) \qquad (2)$$

The sum of the $\vec{l}$ terms, although unknown, would remain constant during the recording of the lead (neglecting changes in conductivity of the body with respiration and pulsatile blood flow,[4-5] and the change in position of the heart with contraction). Therefore the recorded difference in potential, $P - P'$, would fluctuate in direct proportion to variations in the equivalent dipole, $\vec{E}$, at each instant of time. This is the ultimate goal of vectorcardiographic recording, but it is apparent that all of the electromotive forces generated in the heart would never possess the same magnitude and direction so that this approach must be abandoned.

2. If an electrocardiographic lead could be developed so that

$$\vec{l_1} = \vec{l_2} = \ldots = \vec{l_n} = \vec{l},$$

Equation (1) could be rewritten as

$$P - P' = \vec{l} (\vec{e_1} + \vec{e_2} + \ldots \vec{e_n}). \qquad (3)$$

Again neglecting mechanical displacement of the heart during systole and diastole and the effects of respiration and pulsatile blood flow on conductivity, $\vec{l}$ could be considered to remain constant. The fluctuations in $P - P'$ would be directly proportional to the sum total of the variations of each of the individual dipoles. Again the utopia of the vectorcardiographer would be attained. It is not immediately apparent, however, whether the assumption concerning equality of the $\vec{l}$ terms is within the realm of practical achievement. It is the purpose of this communication to report investigation of one facet of this problem.

GENERAL METHODS

A circular rather than a spherical homogeneous medium is postulated in order that the concepts involved may be more simply presented in two-dimensional illustrations. The "heart" is also assumed to have a circular form with a radius of one arbitrary unit. A dipole is located at the center of the "heart" and six other dipoles are located on its surface. Two separate circular "body surfaces" are postulated, the smaller having a radius of two units and the larger having a radius of four units. As illustrated in Fig. 1, the centers of these three circles are assumed to coincide. The dipole at the center of the "heart" is, therefore, centric but the other six dipoles are eccentric with respect to the "body surfaces." The equations developed by Wilson and Bayley[6] are used for all of the calculations, Equation (13) of their paper serving for the centric dipole and Equation (19) for the eccentric ones. In both equations,

R = 2 for the smaller "body surface" and 4 for the larger "body surface." In Equation (19), f = 0.5 for the smaller "body surface" and 0.25 for the larger one. In both equations M = 1. The other terms in these equations are defined in the paper of Wilson and Bayley and, of course, are dependent upon the locations of the points on the "body surfaces" at which potentials are to be determined. For the purposes of this study, these points are chosen at 5° intervals about the entire circumference of the smaller "body surface" and at 0°, 90°, 180°, and 270° of the larger "body surface." The third group of terms in Equation (19) for the z component is omitted since the calculations are being made for a circular rather than for a spherical medium.

Fig. 1.—The "heart" is represented by the inner circle, having a radius of one unit. The locations of the seven dipoles of the "heart" are depicted by dots enclosed in circles. The smaller "body surface" is represented by the intermediate circle, having a radius of two units. The outer circle with a radius of four units represents the larger "body surface." See text.

GRAPHIC METHODS AND RESULTS

The dipoles on the "heart surface" are located at the positions illustrated in Fig. 1 by means of dots enclosed in circles. In order to construct a lead vector between two points of the body surface for any given dipole, the voltage at each point is calculated for the x component when the dipole axis is oriented horizontally and for the y component when the dipole axis is oriented vertically. The principles underlying such lead vector constructions are fully described elsewhere.[1]

The lead vectors between points A and A′ of the smaller "body surface" are illustrated for each of the seven "heart" dipoles in Fig. 2. It is to be noted that the magnitudes and directions of these vectors are substantially different

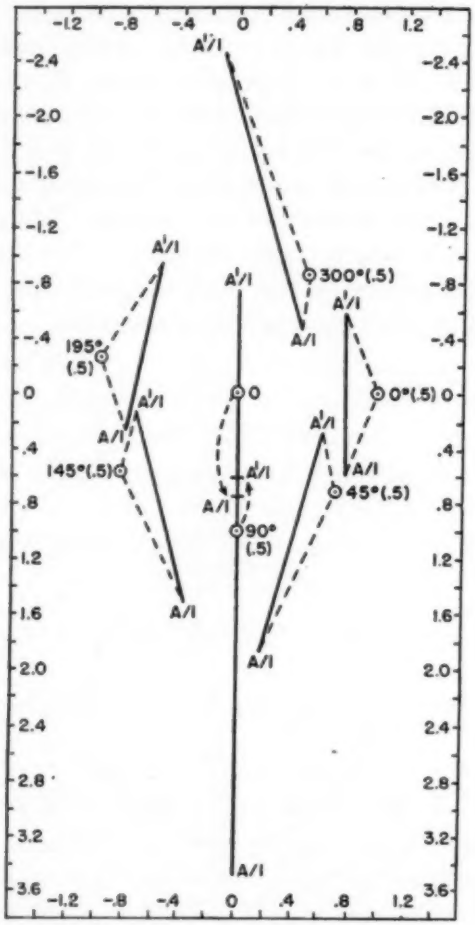

Fig. 2.—The seven lead vectors of lead A/1 — A′/1. See text. (In this illustration, as well as in Figs. 3-6, only the magnitudes and directional axes of the lead vectors are shown. An arrow-head depicting the sense of each vector is omitted, since the polarity of a lead is arbitrary and dependent upon the galvanometer connections. The scales of Figs. 2 to 6 are identical and are based on voltages calculated from the equations of Wilson and Bayley[6] for dipole moments of unity.)

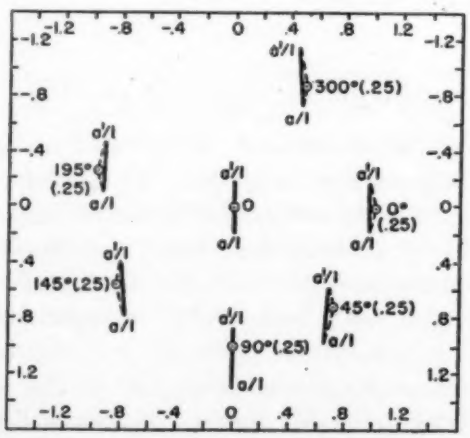

Fig. 3.—The seven lead vectors of Lead a/1 — a′1. See text.

so that they cannot be considered to approach equality. Similarly, the lead vectors between points a and a′ of the larger body surface are illustrated in Fig. 3. The magnitudes of these vectors are much smaller than those of Fig. 2 due to the fact that the points on the larger "body surface" are relatively remote from the "heart." However, the magnitudes and directions of these vectors demonstrate much less variation among themselves. From their appearance one gains the impression that these vectors might be sufficiently similar to justify the use of Equation (3) given previously.

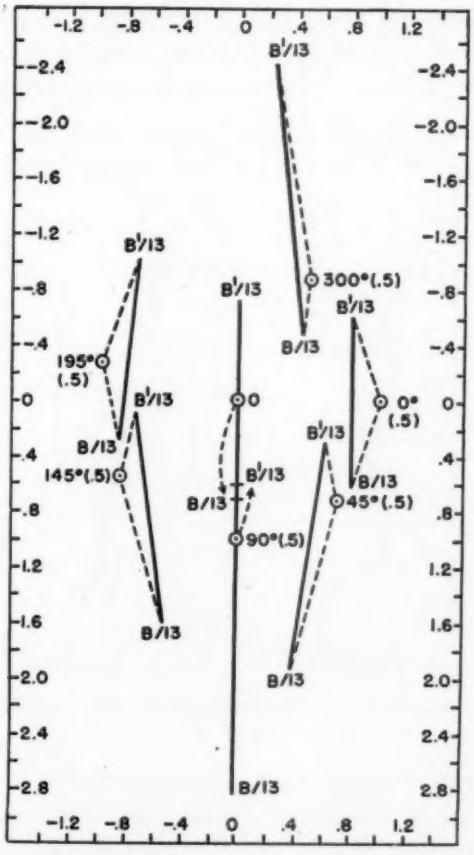

Fig. 4.—The seven lead vectors of Lead B/13 — B′/13. See text.

Is it possible to devise leads for the smaller "body surface" which would approximate the ideal conditions required by Equation (3)? McFee and Johnston and Reynolds and associates have recently suggested[2,7] that, from a vectorcardiographic standpoint, a more satisfactory anterior-posterior lead could be obtained if one galvanometer terminal were joined through equal resistors to multiple points on the precordium and the other terminal through equal resistors to similar multiple points on the back. In order to evaluate this concept the voltages of the thirteen points from 60° through 120° (at 5° intervals) were calculated and averaged for each of the 7 dipoles, each oriented at first horizontally and then vertically. Similarly, the corresponding voltages of the

thirteen points from 240° through 300° were determined and averaged. It should be noted from Fig. 1 that, if lines were drawn between the 60° and 300° points and between the 120° and 240° points, they would form tangents with the "heart surface" circle. In Fig. 1 the arc from 60° to 120° is designated as B and the arc from 240° to 300° as B′. The average voltages of the thirteen points on these arcs are designated as B/13 and B′/13, respectively. The lead vectors of the lead recording the difference in potential between B/13 and B′/13 are illustrated in Fig. 4 for each of the seven dipoles. It may be noted, upon compairson with lead A/1 - A′/1 of Fig. 2, that the lead vectors assume a somewhat more uniform magnitude and are oriented in a somewhat more uniform direction. However, from a qualitative standpoint, the degree of improvement of lead B/13 - B′/13 over lead A/1 - A′/1 is not particularly striking.

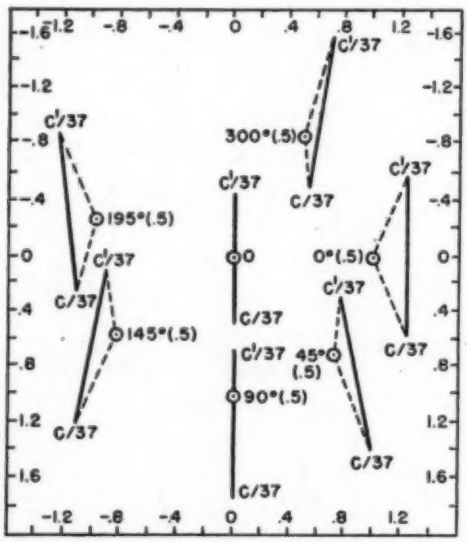

Fig. 5.—The seven lead vectors of Lead C/37 — C′/37. See text.

McFee and Johnston[2] state that, to make a lead field exactly equal to a desired value, it would be necessary to have an infinite number of resistors attached to an infinite number of minute electrodes covering the entire body surface. It was decided, therefore, to study an approximation of such an "ideal" lead. In order to cover the entire "body surface," the arcs from 0° to 180° and from 180° to 360° were chosen; these are designated in Fig. 1 as C and C′, respectively. Rather than averaging the voltages of an infinite number of points on these two arcs, the voltages of the thirty-seven points located at 5° intervals along each arc were calculated and averaged for each of the seven dipoles. This lead is, therefore, designated as C/37 - C′/37; its seven component lead vectors are illustrated in Fig. 5. These vectors have very similar magnitudes, but the reader's attention is drawn especially to their peculiar orientation. The lead vectors are not oriented in parallel directions as might be expected for an ideal lead, predicted on the basis of the lead field concept.[2] Rather the lead vectors tend to diverge from each other at their more peripheral ends. Serial exami-

nation of Figs. 2, 4, and 5 indicates that while the lead vectors of lead B/13 - B′/13 are somewhat more parallel than those of lead A/1 - A′/1, the lead vectors of lead C/37 - C′/37 have "overshot," so to speak, the ideal situation. These observations suggested to the author that an ideal lead need not be formed from multiple electrode points distributed over the entire body surface. It was decided, therefore, to test the possibility that arcs of lengths intermediate between B and C and between B′ and C′, respectively, might be more suitable for developing a more nearly ideal lead in the circular homogeneous model. After calculating lead vectors from arcs of various lengths, it was finally demonstrated by the statistical methods described in the next section that, under the conditions defined of using points at 5° intervals, the best possible lead could be obtained by averaging such points on the arcs extending from 30° to 150° and from 210° to 330°. These arcs are labeled as D and D′, respectively, in Fig. 1 and, since twenty-five points were averaged on each arc, the lead so constructed

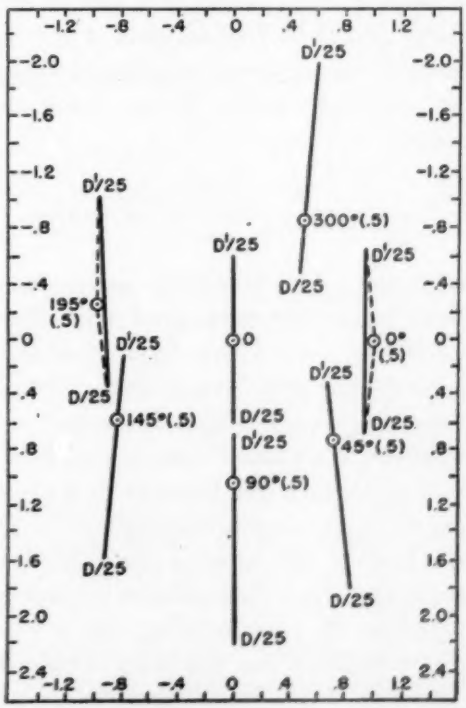

Fig. 6.—The seven lead vectors of Lead D/25 — D′/25. See text.

is designated as D/25 - D′/25. The lead vectors of this lead are illustrated in Fig. 6. While their magnitudes vary to a somewhat greater extent than those of the lead vectors shown in Fig. 5 for lead C/37 - C′/37, these vectors are very nearly parallel. Lead D/25 - D′/25 is, therefore, an almost "perfect" vectorcardiographic lead under the conditions dictated by the use of a circular homogeneous model. It should be noted in passing that the dipoles in Fig. 6 seem to be located upon their respective lead vectors except in the case of those more laterally placed. This visual impression is not mathematically valid, however,

since calculation of the positions of the lead vectors with a degree of accuracy greater than that which can be depicted in the illustrations indicates that, as the "midline" is approached, dipole and vector approach coincidence, but that the latter is not absolute except in the case of the centric dipole and the 90° dipole.

STATISTICAL METHODS AND RESULTS

It would be desirable to be able to express quantitatively the relative degree of variation of an actual lead from an ideal lead. Such an expression not only would be useful for the problem at hand, but also would have a wide field of application in the study of electrocardiographic leads in three-dimensional models.[8-10] For this reason the method of deriving a suitable statistical expression to evaluate not only planar but also spatial vectorcardiographic leads will be discussed in some detail.

In a "perfect" vectorcardiographic lead, the individual lead vectors for each dipole of the heart must be identical. With this fact in mind, it is evident that the variances of the x, the y, and the z components of the individual lead vectors about their means represent the appropriate statistical expressions for evaluating the degree of accuracy of an actual lead. These three variances may then be pooled to obtain the quantity,

$$\frac{S(X - \bar{x})^2 + S(Y - \bar{y})^2 + S(Z - \bar{z})^2}{(3n - 3)},$$

written in accordance with the usual symbolic nomenclature.[11] However, the pooled variance of one lead cannot be compared with that of another lead because the two leads would not necessarily have similar average magnitudes. To illustrate this point the reader may recall that lead D/25 - D′/25 (Fig. 6) has an average lead vector magnitude which is much larger than that of lead a/1 - a′/1 (Fig. 3). Therefore, the pooled variance of lead D/25 - D′/25 might be larger than that of lead a/1 - a′/1 not because of a greater error involved in the former but because of its larger average magnitude. Division of the pooled variance of a lead by the square of the mean of the magnitudes of its lead vectors introduces the necessary correction. This correction, therefore, can be obtained by dividing the pooled variance by the quantity, $(\bar{x}^2 + \bar{y}^2 + \bar{z}^2)$. The resulting expression may be further simplified for use with a calculator. Written in its most convenient form, the final equation, expressing in per cent the coefficient of variation, C, of an actual lead from an ideal lead, is

$$C = \sqrt{\frac{10^4 n}{(3n - 3)}\left(\frac{n(SX^2 + SY^2 + SZ^2)}{(SX)^2 + (SY)^2 + (SZ)^2} - 1\right)} \qquad (4)$$

where n represents the number of dipoles under consideration, SX^2 is the sum of the squares of the x components of the n lead vectors, $(SX)^2$ is the square of the sum of the x components of the n lead vectors, and the Y and Z terms have definitions corresponding to those of the X terms. The degrees of freedom are (3n − 3). For two-dimensional data, such as that developed in this study, the Z terms are omitted and the degrees of freedom are (2n − 2) which replaces

the corresponding term in the denominator of Equation (4). Omission of the radical yields the quantity, 10^4C^2, which may be used with the corresponding quantity of another lead to form a variance ratio for determining the probability that one lead is more nearly ideal, for vectorcardiographic purposes, than the other. For the twelve degrees of freedom resulting from the use of seven dipoles in the present study, the variance ratios corresponding to the 10 per cent, 5 per cent, 2 per cent, and 0.2 per cent levels of probability are 2.687, 3.277, 4.155, and 7.000, respectively.*

TABLE I. THE MEAN MAGNITUDE ($\bar{1}'$) AND COEFFICIENT OF VARIATION (C) OF THE SEVEN LEAD VECTORS OF VARIOUS LEADS CALCULATED FOR THE MODEL ILLUSTRATED IN FIGURE 1

NO.	LEAD	$\bar{1}'$ (IN ARBITRARY UNITS)	C (IN %)	C^2 ($\times 10^4$)
1	A/1–A′/1	1.68	28.9	835.3
2	a/1–a′/1	0.38	6.6	43.7
3	B/13–B′/13	1.60	17.9	321.4
4	C/37–C′/37	1.08	11.5	131.8
5	D/25–D′/25	1.41	7.7	58.9
6	C/13–C′/13	0.97	18.7	348.2
7	D/3–D′/3	1.18	17.5	307.8
8	D/5–D′/5	1.33	10.8	116.6
9	D/9–D′/9	1.36	8.4	70.4
10	A/1–0	0.98	56.6	3201.9
11	A′/1–0	0.70	52.4	2741.1
12	a/1–0	0.21	23.1	532.6
13	a′/1–0	0.18	22.7	516.2
14	D/25–0	0.77	27.2	739.1
15	D′/25–0	0.63	29.6	877.3

In Table I the column designated by the symbol, $\bar{1}'$, includes the mean of the magnitudes of the lead vectors of the leads under consideration. Accordingly, $\bar{1}'$ is equivalent to $\frac{\bar{1}_1 + \bar{1}_2 + \cdots \bar{1}_n}{n}$ and is, therefore, to be distinguished from $\bar{1}$ which is equivalent to the magnitude of any one of the lead vectors of a given lead when all are equal. (See previous assumption underlying Equation (3)). It is evident that $\bar{1}$ and $\bar{1}'$ are not identical unless the lead is "perfect" or ideal which is the only condition under which the quantity, $\bar{1}$, can exist. In the column designated by the letter, C, the coefficient of variation of a lead from the ideal situation is expressed in per cent. The values of the square of C are included in the last column for the convenience of the reader. These values permit the variance ratio to be calculated by dividing the larger C^2 value of a lead by the smaller C^2 value of the lead with which the former is to be compared.

*The tabular probabilities are doubled[11] since the calculation of the variance ratio involves the choice of the two variances for numerator and denominator; the larger variance is arbitrarily taken as the numerator in order that the resulting ratio might exceed unity to coincide with the form in which tables of variance ratios are published.

From the variance ratio, the approximate range of probability that chance alone accounts for the fact that one lead demonstrates a greater degree of variation from the ideal situation than demonstrated by the second lead may be deduced from the variance ratios for selected probability levels given in the preceding paragraph.

Table I includes various leads calculated for the dipole locations depicted in Fig. 1. The first five leads listed in Table I correspond, respectively, to the leads illustrated in Figs. 2 through 6. The coefficients of variation confirm in a quantitative manner the qualitative impression that lead A/1 - A′/1 would be a poor vectorcardiographic lead while leads a/1 - a′/1 and D/25 - D′/25 approach the ideal situation much more closely. It is of interest that the value of $\bar{1}'$ of D/25 - D′/25 is relatively close to that of A/1 - A′/1 while $\bar{1}'$ of a/1 - a′/1 is considerably smaller. This means that a relatively low coefficient of variation has been achieved by the use of lead D/25 - D′/25 with very little sacrifice in the size of the deflections which it is capable of recording at a standard sensitivity of the galvanometer; the significance of this observation as it relates to the experimental findings of Johnston and McFee[12] will be commented upon in the discussion.

Table I also presents proof that lead D/25 - D′/25 is numerically superior to lead C/37 - C′/37, recorded from points over the entire "body surface," although this superiority is not statistically significant. On the other hand, lead B/13 - B′/13, formed by 60° arcs on the smaller "body surface" is inferior to lead D/25 - D′/25 to a degree which is highly significant. This indicates that optimum arcs exist for the recording of an almost ideal lead. (It should be mentioned, however, that while arcs D and D′ yielded the smallest value of the coefficient of variation, arcs which were 5° or 10° longer or shorter than these resulted in coefficients of variation which were very similar.)

For any given pair of arcs, does the number of points, the voltages of which are averaged in the formation of a lead, influence the variability of the lead vectors? This question was first investigated by averaging the voltages of thirteen points located at 15° (rather than at 5°) intervals on arc C and the voltages of the thirteen corresponding points on arc C′. This lead is designated as C/13 - C′/13 to distinguish it from C/37 - C′/37. From Table I it may be seen that the former lead is inferior to the latter at approximately the 10 per cent level of probability. The seventh, eighth, and ninth leads in conjunction with the fifth lead listed in Table I cast additional light on the question of the importance of the number of points selected. Lead D/3 - D′/3 represents the difference between the average potential of three points at 30°, 90°, and 150° on arc D and the average potential of the three corresponding opposite points on arc D′. Thus, these points are chosen at 60° intervals. In lead D/5 - D′/5, five points are selected at 30° intervals on the D and D′ arcs by including points at 60° and 120° on the former and points at 240° and 300° on the latter. In lead D/9 - D′/9 an additional four points are selected on each arc so that this lead consists of the difference between the average voltages of nine points taken at 15° intervals on each of the two arcs. Table I indicates that the lead vectors of these leads become more uniform with an increasing number of points, but

that this greater degree of uniformity approaches a plateau so that there is very little difference between lead D/9 - D′/9 and lead D/25 - D′/25. The significance of these findings with respect to the development of simple and practical vectorcardiographic leads will be discussed later.

The remaining leads listed in Table I are "unipolar." The reference point of each of the lead vectors of such a lead is arbitrarily assigned a value of zero. Such a concept is entirely theoretical since it would be impossible to select an "indifferent" point or network of points which would have a potential equivalent to the mid-potential of all of the dipoles within the heart acting simultaneously. However, it is of interest that the coefficients of variation of the unipolar leads are considerably larger than the bipolar lead formed by the "exploring" electrodes of two unipolar leads.

Fig. 7.—In contrast to Fig. 1, the seven dipoles are grouped into a single quadrant of the inner circle in order that the "heart" and the smaller "body surface" (intermediate circle) might simulate more closely a transverse section of the human thorax at ventricular level. The larger "body surface" (outer circle) is included to permit the formation of remote or distant leads. See text.

In Fig. 7 the dipoles are so arranged as to be included within only one quadrant of the circle representing the heart. In effect this reduces the size of the cluster of dipoles representing the heart. However, the dipole cluster is now eccentrically placed so that it retains its former proximity to a portion of the "body surface" but achieves a more remote position with respect to other portions of the "body surface." Fig. 7 could then be taken as a crude representation of the cross section of the thorax where points A and E are located on the mid-sternal and left mid-axillary lines, respectively, and points A′ and E′ are placed on the vertebral and right mid-axillary lines, respectively. The larger outer circle represents a "body surface" which is disproportionately large for the "heart." The inner "body surface" simulates anatomically the trans-

verse cross section of the human body at ventricular level much more closely than the outer "body surface." The latter is included in this analysis to provide some means of comparing the accuracy of leads recorded by various means on the thorax with leads recorded from more remote positions of the body. For example, although a circle is admittedly an extremely inaccurate representation of the contour of a frontal section of the body, the points a and a′ might be considered to represent the left leg and neck, respectively. Then lead a/1 - a′/1 might be taken to represent a lead between these two anatomic sites, and such a lead, which might be classified as "remote," could be compared with a "precordial" lead formed in various ways on the inner circle. On the basis of this and other analagous considerations the inclusion of two "body surfaces" in the mathematical model seemed to be warranted.

The statistical analysis of leads calculated on the basis of the dipole arrangement illustrated in Fig. 7 is summarized in Table II. The symbols used to represent the leads are identical with, or analagous to, those defined previously. Thus a letter refers to either a point or an arc illustrated in Fig. 7. If the letter refers to a point, the voltage of the point is represented by the letter in the numerator and the number "1" in the denominator. If the letter refers to an arc the average voltage of a number of points on the arc is represented by the letter in the numerator and the number in the denominator corresponding to the number of points at which potentials were calculated and averaged. In the case of an arc the points chosen are located at equally spaced intervals. In Table II, certain leads are formed by assuming a network joining, through equal resistors, either two points (leads 12, 17, 20, 23, 24, 25, 36, 37, 38, 39) or the two mean potentials of the points located on two arcs (leads 14, 19).

In Table II, as in Table I, the mean magnitude of the lead vectors of each lead is listed as well as the coefficient of variation of the lead vectors and its square. The leads are listed in the order of their increasing coefficients of variation for the smaller "body surface" (upper portion of table) and in a like manner for the larger "body surface" (lower portion of table). For convenience a lead will be referred to by its designated number rather than by its symbol.

Certain salient facts to be gleaned from the data presented in Table II are the following:

1. The lead vectors of bipolar leads formed by averaging the voltages of points on arcs of optimal length (leads 1 and 2) are much more uniform in magnitude and direction than are those of similar bipolar leads between single points (leads 9 and 11).

2. There is very little difference in the coefficient of variation of leads 1, 2, and 3 in which one electrode is attached to a large number of points on an arc in proximity to the "heart" while the other electrode is attached to either a single point or a number of points on arcs of varying length more distant from the "heart." On the other hand, if a "point electrode" is substituted for an "arc electrode" in proximity to the "heart" (lead 10), significant deterioration of lead vector uniformity occurs.

3. Leads formed by electrodes at diametrically opposite points (leads 9, 11) demonstrate more uniformity of their component lead vectors than leads

between points enclosing only a quadrant of the circumference of the circular medium (leads 13, 15, 18, 22). In the case of the larger "body surface" this is even more strikingly demonstrated when leads 26 and 27 are compared with leads 32, 33, 34, and 35.

4. By the use of "arc electrodes" it is possible to obtain leads on the smaller "body surface" (leads 1, 2, 3) which are entirely comparable in lead vector uniformity to the best leads obtainable on the larger "body surface" with "point

TABLE II. THE MEAN MAGNITUDE ($\bar{1}'$) AND COEFFICIENT OF VARIATION (C) OF THE SEVEN LEAD VECTORS OF VARIOUS LEADS CALCULATED FOR THE MODEL ILLUSTRATED IN FIGURE 7

NO.	LEAD	$\bar{1}'$ (IN ARBITRARY UNITS)	C (IN %)	C^2 ($\times 10^4$)
1	D/25—D′/25	1.41	6.7	45.5
2	D/25—B′/13	1.42	6.7	45.5
3	D/25—A′/1	1.42	7.2	51.3
4	D′/25—0	0.50	16.3	266.0
5	B/13—B′/13	1.60	17.1	292.8
6	D/25—0	0.91	17.9	320.2
7	A′/1—0	0.51	21.4	459.7
8	E′/1—0	0.49	22.5	505.2
9	E/1—E′/1	1.85	26.4	698.8
10	A/1—D′/25	1.69	26.5	704.1
11	A/1—A′/1	1.69	27.1	736.7
12	D/25—½ (E/1 + E′/1)	1.18	33.8	1145.1
13	A/1—E/1	2.23	34.8	1208.7
14	E′/1—½ (D/25 + D′/25)	0.57	34.9	1219.2
15	A′/1—E′/1	0.58	35.1	1234.3
16	E/1—0	1.39	39.5	1556.9
17	E/1—½ (A/1 + A′/1)	1.67	40.1	1608.8
18	A′/1—E/1	1.45	40.9	1671.8
19	E/1—½ (D/25 + D′/25)	1.46	41.0	1678.8
20	A/1—½ (E/1 + E′/1)	1.56	41.3	1709.7
21	A/1—0	1.21	42.0	1763.4
22	A/1—E′/1	1.26	46.8	2185.7
23	D′/25—½ (E/1 + E′/1)	0.55	64.2	4118.3
24	A′/1—½ (E/1 + E′/1)	0.61	65.7	4320.7
25	E′/1—½ (A/1 + A′/1)	0.52	73.4	5380.1
26	e/1—e′/1	0.39	6.0	36.2
27	a/1—a′/1	0.38	6.1	36.7
28	a′/1—0	0.15	11.9	141.2
29	e′/1—0	0.15	13.0	169.7
30	e/1—0	0.24	15.8	250.2
31	a/1—0	0.23	16.0	254.9
32	a/1—e/1	0.37	17.9	321.4
33	a′/1—e/1	0.29	18.6	347.1
34	a′/1—e′/1	0.19	18.7	350.6
35	a/1—e′/1	0.27	19.8	393.2
36	e/1—½ (a/1 + a′/1)	0.27	21.9	478.9
37	a/1—½ (e/1 + e′/1)	0.26	22.9	522.7
38	a′/1—½ (e/1 + e′/1)	0.15	29.9	896.0
39	e′/1—½ (a/1 + a′/1)	0.14	32.5	1054.1
40*	A/1—0	0.82	54.3	2951.0
41*	a/1—0	0.19	23.2	536.0

*Based on twenty-one lead vectors resulting from the placement of fourteen additional dipoles on the remaining quadrants of the circumference of the "heart" in locations corresponding to those of the dipoles illustrated in the single quadrant of Figure 7.

electrodes" (leads 26, 27). The former type of lead, however, retains, in large measure, the ability to produce, at a given galvanometer sensitivity, the large electrocardiographic deflections characteristic of semidirect leads (see column $\bar{1}'$).

5. "Unipolar" leads in which the voltage of the indifferent point is arbitrarily considered to be zero (leads 4, 6, 7, 8, 16, 21, 28, 29, 30, 31) demonstrate, in almost all instances, a greater degree of uniformity of their lead vectors than the corresponding leads in which the voltage of the "indifferent" point is that of a central terminal of a network of two diametrically opposite points joined through equal resistors (leads 23, 12, 24, 25, 17, 20, 38, 39, 36, 37, respectively).

6. The average voltage of two diametrically opposite "arc electrodes" serves as a better central terminal for an "exploring" electrode relatively distant from the heart than does a central terminal formed by the average voltage of two diametrically opposite "point electrodes." (Compare leads 14 and 25.) However, in the case of an "exploring" electrode located relatively close to the heart, the choice of an "indifferent" reference potential does not influence significantly the coefficients of variation calculated. (Compare leads 16, 17, and 19 and also leads 20 and 21.)

7. In general, bipolar leads between two points are superior to "unipolar" leads between a point and a central terminal, both with the smaller and the larger "body surfaces"; this is particularly evident from Table II in the case of the latter. This finding is undoubtedly partially attributable to the fact that one arm of the central terminal is attached to a point which is relatively close to the "heart." Unsuccessful attempts were made to improve the various central terminals by assuming unequal resistors in their two arms. The results of these negative studies were omitted from Table II in order not to obscure the more pertinent findings with excessive detail.

8. The data for leads 40 and 41 were calculated from Equation (4) after increasing the number of lead vectors to twenty-one by including additional dipoles on the other three quadrants of the circumference of the "heart" in locations corresponding to those illustrated in the single quadrant of Fig. 7. The coefficient of variation of these twenty-one lead vectors for point A, located one heart diameter from the center of the "heart," is 54.3 per cent; for point a, located two heart diameters from the center of the heart, this coefficient falls to 23.2 per cent. These values are numerically very similar to those reported by McFee and Johnston,[2] based on a different method of calculation for a random distribution of electromotive forces throughout the heart. The use of Equation (4), however, makes the assumption of a random distribution unnecessary. Thus, when only seven dipoles are confined to a single quadrant, the coefficients of variation may be stated to be 42.0 per cent and 16.0 per cent for points A and a, respectively. (See leads 21 and 31.)

DISCUSSION

Most vectorcardiographic reference frames, such as the various forms of the tetrahedron and the rectilinear trihedron, are based upon the assumptions that the heart functions electrically as a single fixed equivalent dipole, located at equal distances from the several electrode attachments in a medium which is

not only homogeneous but also either spherical or infinite. In recent years, increasing attention has been given to eliminating certain of these assumptions through the use of the concept of the lead vector.[10] It is now possible to calculate for any point on or within a homogeneous sphere, the potential developed by a dipole located anywhere within the sphere.[6] Thus the assumption of a centric dipole is no longer a necessity. Furthermore, by using the concept of the lead vector in conjunction with experimental studies carried out on models[10] or cadavers,[13] it is possible to eliminate the necessity of assuming a homogeneous spherical medium. However, the lead vector concept does not obviate the necessity of assuming a single fixed equivalent dipole for the heart. Much work has been done in an attempt to evaluate the relative accuracy of the latter assumption, but at the present time it remains controversial. Reluctance to accept the assumption of a single fixed equivalent dipole has led to the introduction of alternative concepts such as the "lead field" of McFee and Johnston[2] and the "tubes of influence" of Lepeschkin.[14] For the same reason the closely related concept of multiple lead vectors was recently suggested by the author.[1] In the present study the asumptions concerning the boundary configuration and the homogeneity of the medium were retained in order to allow the necessary calculations to be made from the available equations. However, it should be emphasized that the methods used herein might well be applied to the use of multiple rather than single dipoles located within the heart of a cadaver or within the heart region of a model. It would then be possible to eliminate the assumptions concerning the conducting medium.

In the author's experience it has been difficult to predict with accuracy the lead field of a conducting medium. Thus, with the technique of field mapping[14] it would have seemed likely that lead C/37 - C′/37 should have produced a more uniform field on the circular homogeneous lamina than that formed by lead D/25 - D′/25. It should be emphasized, however, that the concepts of the lead field and of multiple lead vectors are fundamentally similar and equally valid. The use of fluid mappers[15] or conducting paper[16] would have led to the same conclusions as the calculations herein presented.

The calculation of the lead vectors for multiple dipoles proves conclusively, at least for a circular homogeneous lamina, the validity of the suggestion[2] that a bipolar lead formed by multiple electrodes connected through equal resistors to each of the poles of a galvanometer produces a better vectorcardiographic lead than could be achieved with a bipolar lead between two electrodes. In a recent report,[12] evidence was presented that leads recorded between two networks of points, one on the precordium and one on the back, yielded deflections which were considerably larger than those obtained with a "remote" unipolar lead (such as V_B) recorded along an axis having a similar average direction. It might have been supposed that if points over a sufficiently large portion of the precordium were averaged, by means of a network, to produce a relatively uniform field, the resulting voltage would be greatly reduced, and, conversely, that the recording of potential differences not much smaller than those obtained with the usual precordial leads would indicate very little improvement over the lead fields of the latter. The present study, however, demonstrates conclusively

that it is theoretically possible to obtain a relatively uniform lead field with very little sacrifice in recorded voltage, and, therefore, lends support to the contention of Johnston and associates[2,7,12] that their sagittal lead is superior to those currently used for vectorcardiographic purposes.

It has been suggested[7] that a network of points joined through equal resistors could be replaced by flexible metal plates or a metal mesh to make such leads practical for routine use. In the author's opinion a simplification in technique could be achieved by constructing the electrodes of stainless steel foil of the type used by Lepeschkin.[17] Such an electrode could be cut to any desired size and shape and made to adhere uniformly to the chest surface by means of electrode paste. Based upon leads 6, 7, 8, and 9 of Table I, the use of such an electrode would have an advantage over joining a relatively small number of electrodes through equal resistors and would probably be equivalent to joining a relatively large number of such electrodes.

If the results of the calculations given in Table II for the homogeneous circular lamina can be carried over to the recording of a clinical vectorcardiogram, the following scheme is suggested as a more rational reference frame than those currently in use:

1. Longitudinal lead between a point on the left leg and the left side of the neck (analogous to leads 26 and 27 of Table II).

2. Sagittal lead between a large stainless steel foil electrode centering on the V_2 position and a similar electrode centering on a corresponding opposite point on the back (analogous to lead 1 of Table II). On the basis of leads 2 and 3 of Table II it is likely that the size of the back electrode is not critical.

3. Transverse lead between a large stainless steel foil electrode centering on the V_6 position and a similar electrode centering on the V_{6R} position (analogous to lead 1 of Table II). Again the size of the V_{6R} electrode is probably not critical.

Such a lead system would seem to be superior to the cube frame, the leads of which are analogous at best to leads 32 through 35 (Table II). Likewise, the tetrahedron suffers from making use of standard lead 1 for the transverse component which is more nearly like leads 32 through 35 than like leads 26 and 27. The tetrahedron also depends upon V_B for the sagittal component and V_F for the longitudinal component which, even assuming a "perfect" central terminal, are at best analogous to leads 28 and 29.

The validity of a lead system based upon the placement of large electrodes on the thorax could be investigated in various ways. The voltages of points located on the surface of a model[8,9] in areas corresponding to the sites of the large electrodes could be determined and averaged for a dipole placed successively in a large number of locations of the heart region. The x, y, and z values of each of the lead vectors so determined, i.e., one lead vector for each successive dipole location, could then be substituted in Equation (4) in order to determine the coefficient of variation of the lead. In this manner the optimal size (and perhaps shape) of the large electrodes could be determined. A more refined method of obtaining similar data for substitution in Equation (4) would consist of determining, for various dipole locations, the potential differences between

foil electrodes of various sizes and shapes, molded to conform to the inner surface of the model in the desired areas. These considerations serve to emphasize the versatility of Equation (4) in its application to the analysis of data which has already been collected[8,9] or which can be readily obtained in the future by those working with models.

Another method of studying the validity of the proposed reference frame is that which compares the electrocardiographic deflections of a lead derived from the three component leads of the frame with the deflections of a fourth lead having a similar average axis. The rationale and details of this method, which takes into account the diminution in effective size of the dipole cluster of the heart as the result of synchronous activation of the two ventricles, have been described elsewhere.[1]

It should be emphasized that the suggested reference frame is based on data calculated with the assumption of a circular homogeneous lamina. Whether one is justified in applying such data to a volume conductor as complicated as the human body is open to question. The author is, therefore, not advocating the use of this reference frame in vectorcardiography until its characteristics are further studied by the methods just outlined. Only thus will it be possible to obtain an answer to the question posed earlier concerning the practical application of Equation (3).

SUMMARY

1. The uniformity of the magnitudes and directions of the lead vectors of various leads calculated for multiple dipoles located on an electrically homogeneous circular lamina has been investigated by graphic and by statistical methods.

2. The results of this study strongly support the contention of Johnston and associates that, under certain circumstances, a lead recorded between two networks of points is greatly superior, from a vectorcardiographic standpoint, to a lead recorded between two single points.

3. On the basis of these findings a reference frame is suggested for investigation as to its possible superiority to those currently used in vectorcardiography. Methods of evaluating such a frame are outlined.

4. A statistical expression (Equation 4) involving the concept of the coefficient of variation has been developed to estimate the degree to which a given lead varies from an ideal vectorcardiographic lead. The wide field of application of this expression to the evaluation of data collected from the study of the distribution of dipole currents in models is pointed out.

SUMMARIO IN INTERLINGUA

Esseva investigate le uniformitate del magnitudes e directiones del vectores de varie derivationes, calculate pro multiple dipolos a location super un electricamente homogenee lamina circular. Le resultatos del studio supporta le assertion de Johnston e socios que un derivation registrate inter duo retes de punctas es vectocardiographicamente multo superior a un derivation registrate inter duo punctas individual. Super le base de iste constatationes un carde de

referentia es presentate con le proponimento que illo sia investigate in re su possibile superioritate a illos nunc currentemente usate in investigationes vectocardiographic. Methodos pro le evalutation del cadre es schizzate. Esseva disveloppate un expression statistic, involvente le concepto del coefficiente de variation, pro estimar le grado a que un derivation particular devia ab le derivation vectocardiographic ideal. Es signalate le vaste extension del campo de application de iste expression in evalutar datos colligite ab le studio del distribution de currentes dipolic in modellos e cadaveres.

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A COMPLEX OF CONGENITAL CARDIAC ANOMALIES: VENTRICULAR SEPTAL DEFECT, BIVENTRICULAR ORIGIN OF THE PULMONARY TRUNK, AND SUBAORTIC STENOSIS

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STUDY of hearts that contain congenital ventricular septal defects has made it apparent to us that there is a complex of malformations associated with a ventricular septal defect to which attention should be drawn. This complex is composed of a ventricular septal defect in conjunction with biventricular origin of the pulmonary trunk and subaortic stenosis. In each of the four specimens of this complex that we have seen, aortic obstruction was also present in the region of the aortic arch, manifested as either coarctation or interruption of the aortic arch. The structural features in the specimens that we have studied are sufficiently alike to allow a composite anatomic description of the four cases to be presented, special reference being made to deviations from the usual.

CASE REPORTS

CASE 1.—A 1-month-old boy was admitted to the hospital under the care of physicians of the Mayo Clinic. The complaints were cyanosis with crying for 3 weeks and failure to gain weight. The referring physician had detected cardiac enlargement and had prescribed digitalis.

Examination revealed an anxious expression and rapid respirations; cyanosis was absent when the patient was quiet. Fine râles were heard in both lungs, and the liver was enlarged slightly. The cardiac rate was rapid, and moderately loud systolic and diastolic murmurs were present over the mid-precordium. Pulsations of the femoral arteries were palpable but weak. A roentgenogram of the thorax demonstrated cardiac enlargement and increased pulmonary vascular markings (Fig. 1). Electrocardiograms were interpreted as indicating left ventricular hypertrophy as shown by tall R waves and inverted T waves in Lead V_6.

A clinical diagnosis of congenital heart disease with cardiac failure was made. Patent ductus arteriosus, coarctation of the aorta, and endocardial sclerosis were the possibilities considered, and treatment by administration of oxygen and full digitalization was started immediately in an effort to improve the infant's condition sufficiently for further investigation. A favorable response did not occur, and the baby died 48 hours after admission.

CASE 2.—A girl was born under the care of clinic physicians after a normal gestation and delivery. The weight at birth was 3,320 grams.

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Examination immediately after birth revealed multiple congenital anomalies. A defect was present in the skin over the lumbar portion of the spinal column from which cerebrospinal fluid was oozing; the cranial sutures were separated and the anterior fontanel was tense. Anal sphincteric tone was poor, and the lower extremities were paralyzed. A moderately loud systolic murmur was noted to the left of the sternum in the third intercostal space, and pulsations of the femoral arteries were not palpable. Slight cardiac enlargement, prominence of the pulmonary vascular markings, and a relatively narrow supracardiac shadow were noted on the thoracic roentgenogram. The electrocardiogram was considered to be normal for a newborn.

A clinical diagnosis of congenital heart disease was made, in addition to spina bifida with myelomeningocele and myelodysplasia, and hydrocephalus. The infant became cyanotic. Rapid, grunting respirations and bilateral pulmonary inspiratory râles developed. The baby died 3 days after birth.

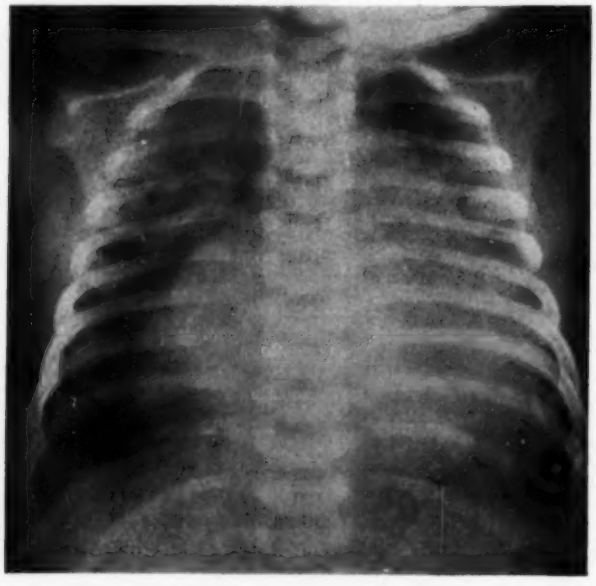

Fig. 1.—Case 1. Thoracic roentgenogram showing cardiac enlargement and prominent vascular markings in the lung.

CASE 3.—An 8-day-old girl was admitted to a hospital elsewhere. Lethargy and poor appetite were noted in the first week of life, with development of grunting respirations and cyanosis. Examination revealed pulmonary inspiratory râles and enlargement of the liver. Cardiac enlargement and pulmonary congestion were noted in the roentgenogram of the thorax. Death occurred within an hour after admission to the hospital. A specimen of the thoracic organs was submitted to us.

CASE 4.—A boy died at 1 week of age; the clinical course had been observed elsewhere. The pathologic specimen was submitted to us.

PATHOLOGIC FEATURES

The essential findings at necropsy were similar in the four cases and will be described together. Details pertaining to each case are given in the accompanying Table I. The great vessels were properly interrelated, and the pulmonary trunk was noticeably wider than the ascending aorta. Muscular ridges measuring from 2 to 5 mm. from above downward were present in the outflow tracts of the left ventricles. The ridge in each case was continuous

with the ventricular septum behind and with the anterior wall of the left ventricle and with the mitral valve in front. Superiorly, the dividing ridge joined the origins of the aorta and the pulmonary trunk. Inferiorly, the ridge had no attachments, the structure projecting into the cavity of the outflow tract of the left ventricle. This ridge divided the outflow tract of the left ventricle into a posterior subaortic portion and an anterior portion (Fig. 2,*a*). The subaortic portion of the outflow tract was narrow, mainly because of the location

TABLE I. PATHOLOGIC FINDINGS IN FOUR CASES OF VENTRICULAR SEPTAL DEFECT WITH BIVENTRICULAR ORIGIN OF THE PULMONARY TRUNK AND SUBAORTIC STENOSIS

	CASES			
	1	2	3	4
Age of patient at death	38 days	3 days	8 days	7 days
Thickness of right ventricle	6*	5	6	7
Thickness of left ventricle	10	5	8	6
Diameter of pulmonary trunk at valve	12	10	12	14
Diameter of aorta at valve	6	5	6	6
Aortic valve	Bicuspid	Bicuspid	Bicuspid	Normal
Diameter of ventricular septal defect	8	7	8	8
Diameter of subaortic tract†	4	4	5	5
Diameter of anterior tract‡	7	7	6	6
Distance from point of subaortic stenosis to aortic valve	5	2	3	4
Diameter of ductus	4	Right = 3 Left = 3	Right = 1.5 Left = 3	2
Aortic obstruction	3.5 at coarctation	Interruption of arch	Interruption of arch	2 at coarctation

*This and all remaining figures in table are in millimeters.

†This is the tract behind the dividing ridge in the left ventricle.

‡This is the tract in front of the dividing ridge in the left ventricle and leading to the ventricular septal defect.

of the dividing ridge (Fig. 2,*b*). Further cause for its narrowness was the fact that the anterior extremity of the medial surface of the anterior mitral leaflet was attached to the dividing ridge. At its upper end, the subaortic portion joined the normally arising aorta.

That portion of the divided outflow tract of the left ventricle which lay anteriorly communicated with the right ventricle by means of a defect in the anterior and muscular portion of the ventricular septum.

When viewed from the right side, this defect was located in the outflow tract of the right ventricle, above the papillary muscle of the conus and in close relation to the pulmonary valve (Fig. 2,*c*). The inferior border of the defect

was concave and was formed by muscular ventricular septal tissue. Its superior border was formed by varying amounts of muscular tissue except at its left upper aspect, where the edge of the defect was formed in each of the cases by pulmonary valvular tissue. In each instance, that portion of the pulmonary valve which was the commissure between the left and the right pulmonary cusps formed part of the upper edge of the defect. In three of the cases, the left pulmo-

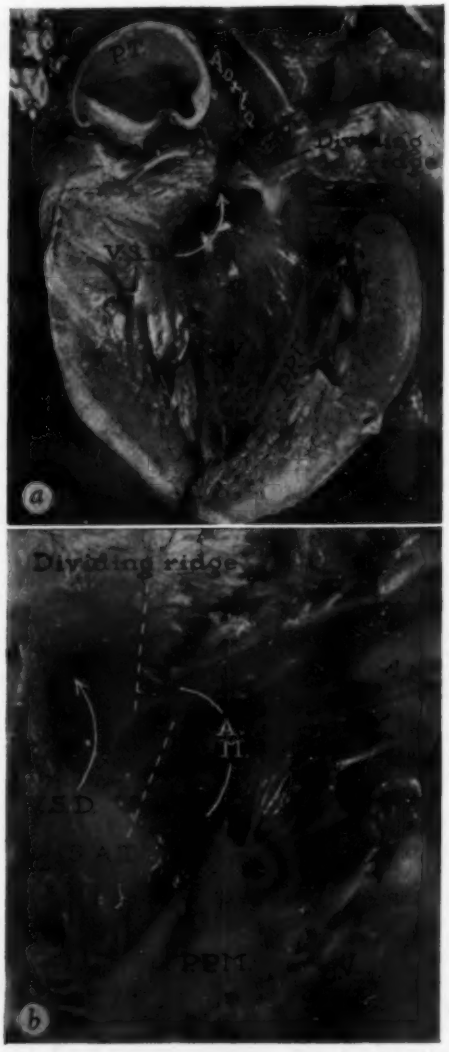

Fig. 2.—Case 1. *a*, The left ventricle (*L.V.*) has been opened in the usual manner. The posterior papillary muscle (*P.P.M.*) and the anterior mitral leaflet (*A.M.*) are normal. Across the outflow tract of the left ventricle is a muscular ridge that divides this outflow tract into two portions, a posterior subaortic portion and an anterior portion that, by way of the ventricular septal defect (*V.S.D.*), leads beyond into the pulmonary trunk (*P.T.*). Note the relatively small size of the ascending aorta compared with the pulmonary trunk. *b*, The unopened outflow tract of the left ventricle is viewed from below upward. The dividing ridge is seen presenting across the outflow tract of the left ventricle. The anterior extremity of the dividing ridge is continuous with the anterior extremity of the anterior mitral leaflet (*A.M.*). The narrowness of the subaortic tract (*S.A.T.*) is the consequence of the position of this dividing ridge and of its relation to the anterior leaflet of the mitral valve. The ventricular septal defect (*V.S.D.*) and the tract leading to it are located anterior to the dividing ridge. *A.P.M.* = anterior papillary muscle.

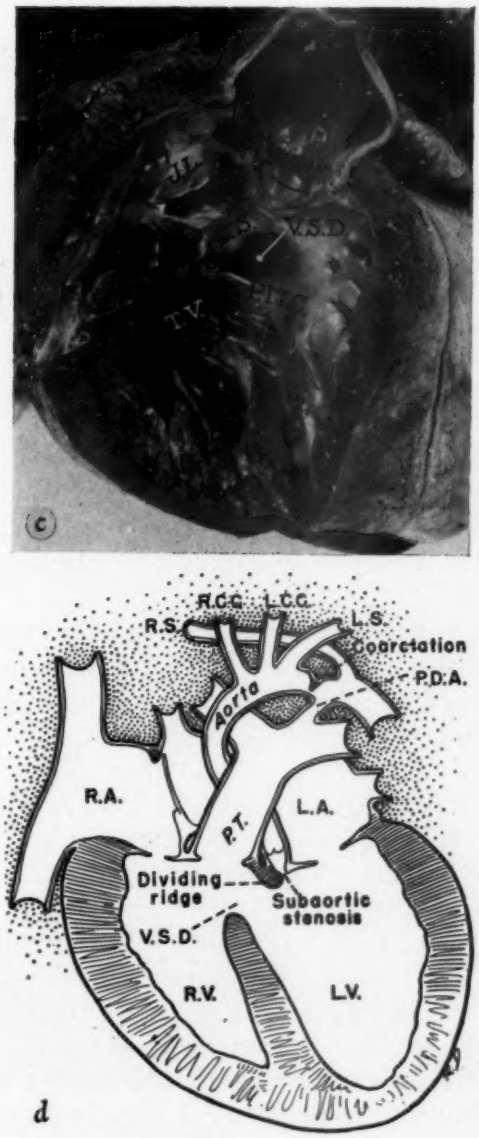

Fig. 2.—Case 1. *c*, The right ventricle (*R.V.*) has been opened in the usual manner. The pulmonary valve (*P.V.*) is wider than normal and overrides the ventricular septal defect (*V.S.D.*). The ventricular septal defect is located above the papillary muscle of the conus (*P.M.C.*) and below the pulmonary valve and a portion of the crista supraventricularis (*C.S.*). On the anterior wall of the outflow tract of the right ventricle is an area of endocardial thickening (*J.L.*) interpreted as a jet lesion resulting from a left-to-right shunt through the defect. *T.V.* = tricuspid valve. *d*, Diagram of the heart and great vessels in this case. The intracardiac features pertain to all four cases. The pulmonary trunk (*P.T.*) is wide and overrides a ventricular septal defect (*V.S.D.*). The subaortic tract is narrowed by the presence of the dividing ridge described in the text, and the ascending aorta is narrow. The first branch of the aortic arch in this case was the right common carotid artery (*R.C.C.*), the second was the left common carotid (*L.C.C.*), and the third was the left subclavian artery (*L.S.*). Beyond the left subclavian artery and proximad to a patent ductus arteriosus (*P.D.A.*) was found coarctation of the aorta. The right subclavian artery (*R.S.*) arose distad to the coarctation and coursed behind the esophagus to the right side of the upper mediastinum.

nary leaflet itself also formed part of the edge of the defect; in one of these, a portion of the anterior pulmonary leaflet likewise contributed to the margin of the defect. The muscular tissue forming varying amounts of the right upper wall of the defect was the crista supraventricularis, which continued toward the left to terminate in the left ventricle as the aforementioned ridge that divided the outflow tract of the left ventricle.

One interpretation of the relations of the defect is that the crista supraventricularis and the pulmonary valve lie above it and the rest of the ventricular septum lies below it. The membranous portion of the ventricular septum was intact and normal in each of the four cases.

The pulmonary trunk and valve were wide. The valve, although intrinsically normal, overrode the ventricular septal defect and so could be said to arise from both ventricles. The degree of overriding was essentially similar in the four cases. The left pulmonary cusp and the left third of the anterior cusp presented over the left ventricle, whereas the right pulmonary cusp and most of the anterior pulmonary cusp overlay the right ventricle. The aorta arose from the subaortic portion of the divided outflow tract of the left ventricle. The aortic valve was bicuspid in three cases. The coronary arteries arose from the aorta.

The heart was noticeably enlarged in each case, and the right ventricular wall was hypertrophied. The left ventricle in Case 1 also was hypertrophied. Both atria and their venous connections were normal. A patent foramen ovale of the valve-competent type was present in each case. The atrioventricular valves were normal.

Malformations of the aortic-arch system were associated with these intracardiac defects in each of the four cases. In Case 1 (Fig. 2,*d*) the ascending aorta was 7 mm. in diameter. The first branch of the aortic arch was not the innominate artery but rather the right common carotid artery, and the second branch was the left common carotid artery. Between the left common carotid artery and the left subclavian artery was noted tubular hypoplasia of the aortic arch, which at this level had a diameter of 5 mm. Distad to the left subclavian artery and proximad to the ductus arteriosus was noted coarctation of the aorta, with characteristic infolding of the aortic media; at the level of the coarctation, the lumen of the aorta was reduced to 3.5 mm. in diameter. The descending aorta beyond the coarctation measured 7 mm. in diameter. A patent ductus arteriosus measuring 4 mm. in diameter was located just distad to the coarctation. The ductus arteriosus arose normally from the left pulmonary artery. The right subclavian artery arose from the posterior wall of the proximal portion of the descending aorta just distad to the coarctation. From its anomalous site of origin, the right subclavian artery coursed behind the esophagus from left to right, slightly compressing this structure, to reach the right side of the superior mediastinum.

Cases 2 and 3 presented identical anomalies of the aortic arch; Case 2 has been previously reported,[1] with emphasis on this aspect. Bilateral persistence of the ductus arteriosus with interruption of the aortic arch was present in each of these cases. The ascending aorta had a normal position and course but

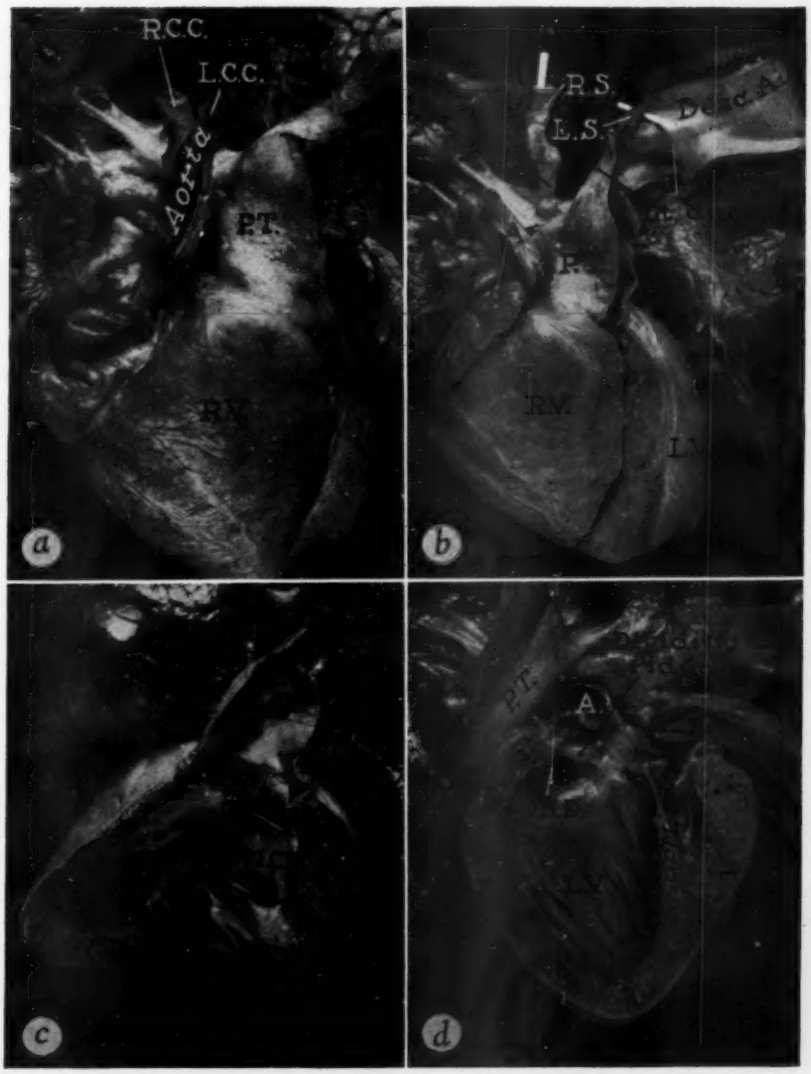

Fig. 3.—Case 2. *a*, The heart and great vessels viewed from in front. The ascending aorta terminates by dividing into the right common carotid (*R.C.C.*) and left common carotid arteries (*L.C.C.*). The pulmonary trunk (*P.T.*) is wide and lies properly related to the aorta. *b*, The ascending aorta (*A*) has been retracted to one side. There is complete interruption of the aortic arch; the descending aorta (*Desc. A.*) originates by connecting with a patent ductus arteriosus (*Lt. ductus*), which arises from the left pulmonary artery. The left subclavian artery (*L.S.*) arises from the descending aorta opposite the junction of the left ductus arteriosus with the descending aorta. On the right side there is also a patent ductus arteriosus (*Rt. ductus*), which arises from the right pulmonary artery and is continuous with the origin of the right subclavian artery (*R.S.*). *c*, The right ventricle and the pulmonary trunk are opened in the usual fashion. Of interest is the location of the ventricular septal defect (*V.S.D.*). It lies above the papillary muscle of the conus (*P.M.C.*) and below the pulmonary valve (*P.V.*) and a portion of the crista supraventricularis (*C.S.*). The pulmonary valve is wide and overrides the ventricular septal defect. *d*, When viewed from the left, the ventricular septal defect (*V.S.D.*) is located anterior to a prominent muscular ridge (*Dividing ridge*), which lies across the outflow tract of the left ventricle. Posterior to this dividing ridge is the narrowed subaortic tract, which continues upward into the aorta (*A*).

terminated by dividing into the right and left common carotid arteries (Fig. 3,*a*). The pulmonary trunk had a normal position and branched normally into the right and left pulmonary arteries. From the left pulmonary artery, a patent ductus arteriosus joined the descending aorta, which was on the left side (Fig. 3,*b*). The left subclavian artery arose from the most proximal portion of the descending aorta opposite the entrance of the left ductus arteriosus. On the right side, a narrow patent ductus arteriosus extended upward from the right pulmonary artery to join the base of the right subclavian artery. The

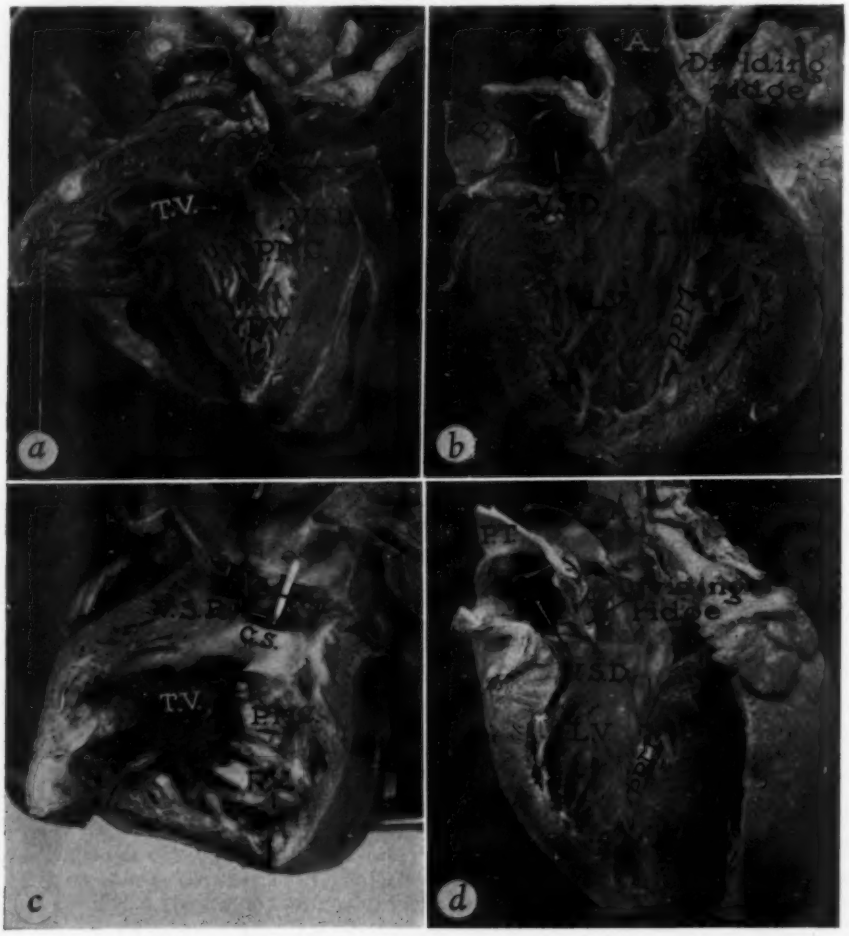

Fig. 4.—The abbreviations are the same as used in the previous figures. In these left ventricular views, the pulmonary trunk has been opened from the left ventricle to show the relations of the ridge dividing the outflow tract of the left ventricle to the root of the aorta and to the pulmonary trunk; thus these views are somewhat different from the corresponding views in Figs. 2 and 3. *a*, Right ventricle, Case 3. *b*, Left ventricle, Case 3. *c*, Right ventricle, Case 4. *d*, Left ventricle, Case 4.

right subclavian artery had no other proximal vascular connections. The intracardiac derangements in this heart are seen in Fig. 3,*c* and *d*. Fig. 4 depicts the findings in Cases 3 and 4.

The heart in Case 4 has been illustrated as Figure 106 of a previous publication.[2] The ascending aorta was thin-walled, and its diameter was 6 mm.

The branches of the aortic arch were normal. Beyond the left common carotid artery and between it and the left subclavian artery was noted tubular hypoplasia of the aortic arch, which measured 3 mm. in diameter along this segment. Coarctation of the aorta, with characteristic infolding of the aortic media, was present beyond the left subclavian artery and proximad to the ductus arteriosus. The lumen of the aorta at this point was reduced to 2 mm. in diameter. A patent ductus arteriosus measuring 2 mm. in diameter was present at its usual location. The descending aorta beyond the ductus arteriosus widened to a diameter of 7 mm.

Microscopic examination of the lungs in all cases revealed massive edema associated with hemorrhage into the alveolar spaces. The pulmonary complications were interpreted as resulting from left ventricular failure and were thought to be the immediate cause of death.

COMMENT

To the best of our knowledge, the combination of anomalies we have described has not been previously reported as a complex. In Taussig's book[3] on congenital malformations of the heart, a case that is similar in all respects to our cases is illustrated as Figure 163 in the chapter on coarctation of the aorta. The author did not make particular comment on the intracardiac anomalies in that case.

The location of the ventricular septal defect in these cases deserves special emphasis in that it did not involve the membranous portion of the ventricular septum but lay anterior to it and above the papillary muscle of the conus. When viewed from the right ventricle, as already mentioned, the defect was seen to open into the distal portion of the outflow tract of the right ventricle. The dividing ridge presenting in the outflow tract of the left ventricle may be interpreted as the left extremity of the crista supraventricularis, and its presence may be interpreted as the cause of the subaortic stenosis.

It is interesting to consider the possible peculiarities of the fetal circulation that might have resulted from this combination of defects. The pulmonary trunk overriding the outflow tract of the left ventricle, coupled with the subaortic stenosis, creates the possibility of a left-to-right shunt at the ventricular level during fetal life. Such a shunt conceivably could reduce the amount of blood that would flow through the ascending aorta and proportionately increase the amount of blood that would flow through the pulmonary trunk. Under these circumstances, it is possible for the pulmonary trunk to dilate and override progressively to a still greater degree the outflow tract of the left ventricle. The possible existence of these hemodynamic features in the fetus would contribute toward comprehension of the basis for the large size of the pulmonary trunk and the relative hypoplasia of the ascending aorta, features that were common to all four patients, even those who were only several days of age at the time of death.

The association of subaortic stenosis and ventricular septal defect constitutes a combination that appears to be particularly disadvantageous for extrauterine life. Since a barrier exists between the ascending aorta and the ven-

tricles, the pressure in the ascending aorta would be less than that in the ventricles. If a normal systemic pressure were to be built up in the proximal part of the aorta, it would require a pronounced increase in pulmonary resistance and arterial pressure. On the other hand, if the ventricular pressure failed to increase to such levels, the pressure in the proximal part of the aorta might be less than that value desirable for proper cerebral circulation, and the relatively low pulmonary vascular resistance would operate to effect a large left-to-right shunt. The latter phenomenon apparently applied in our cases in view of the pulmonary edema and hemorrhage observed at necropsy.

The poor prognosis that was present in our cases of this complex of anomalies is comparable to what has been described in some instances of ventricular septal defect not complicated by the additional anomalies described in this paper.[4-6]

It will be recalled that in each of our cases there were, in addition to the intracardiac malformations, serious obstructive lesions of the aorta in the form of either coarctation or complete interruption of the aortic arch.

The biventricular origin of the pulmonary trunk in the combination of malformations described herein brings up for consideration the so-called Taussig-Bing complex. Biventricular origin of the pulmonary trunk also is present in the latter condition, but important differences exist between the complex here described and the Taussig-Bing complex. In the latter, the great vessels lie in a transposed relationship to each other, and the aorta, lying anteriorly, arises exclusively from the right ventricle, while the pulmonary trunk overrides the ventricular septal defect and lies posteriorly. In the complex here described, the great vessels are not in a transposed relationship to each other, and the aorta arises entirely from the left ventricle.

A situation similar to that present in our cases is to be found in reported examples of corrected transposition with overriding pulmonary trunk. In this latter anomaly, the great vessels are transposed but nevertheless arise from their appropriate ventricles. The aorta is anterior and arises from the systemic ventricle on the left, and the pulmonary trunk is posterior and arises from the venous ventricle on the right. Under these conditions, it has been observed that the pulmonary artery not infrequently overrides a ventricular septal defect.[7] If such is the case, the situation resembles functionally, although not anatomically, the one observed in our cases in that both vessels arise from their appropriate ventricles and at the same time the pulmonary trunk is wide and overrides a ventricular septal defect. The resemblance goes no further, for in corrected transposition with a ventricular septal defect and biventricular origin of the pulmonary trunk, there usually is no subaortic stenosis, although the pulmonary artery is wider than the aorta.

SUMMARY

Four cases are reported that presented an anatomic complex consisting of ventricular septal defect with biventricular origin of the pulmonary trunk and subaortic stenosis.

The ventricular septal defect in this complex is unusual in that it is located anteriorly in the outflow tract of the right ventricle and does not involve the membranous portion of the ventricular septum. The subaortic stenosis is the consequence of a muscular ridge that lies across the outflow tract of the left ventricle and encroaches on the width of the subaortic portion of this outflow tract. The pulmonary artery is not transposed and overrides the ventricular septal defect. Associated anomalies of the aortic arch were present in all cases. Early infantile death from pulmonary hemorrhage and edema occurred in these cases.

SUMMARIO IN INTERLINGUA

Es reportate quatro casos que presentava un complexo anatomic de defecto del septo ventricular combinate con origine biventricular del trunco pulmonar e stenosis subaorotic.

Le defecto septoventricular in iste complexo es inusual in tanto que illo se trova anteriormente in le tracto de effluxo del ventriculo dextere e non involve le portion membranose del septo ventricular. Le stenosis subaortic resulta de un elevation muscular que transversa le tracto de effluxo del ventriculo sinistre e assi reduce le largor de su portion subaortic. Le arteria pulmonar non es transponite e se superimpone al defecto del septo ventricular. Anomalias associate del arco aortic esseva presente in omne casos. In iste casos precoce morte in infantia resultava de hemorrhagia e edema del pulmones.

ADDENDUM

Since preparation of this manuscript, one of us (Edwards) has received from Dr. Edward C. Menefee a specimen from a girl who died suddenly and unexpectedly at the age of 2 weeks.

The intracardiac defects in this instance were identical to those in the four cases described in the main body of this paper. Also, significant malformations were present in the aortic-arch system, in which the pattern was essentially a mirror image of that in Cases 2 and 3. The narrow aorta terminated as the right and left common carotid arteries.

A ductus arteriosus was present on each side. The one arising from the left pulmonary artery was narrow and the left subclavian artery took origin from its upper end. Arising from the right pulmonary artery was a wide patent ductus arteriosus that terminated by being continuous with the descending aorta. The latter vessel lay on the right side and to the right of the esophagus. The right subclavian artery arose from the upper end of the most proximal portion of the descending aorta.

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INCIDENCE OF ASYMPTOMATIC, ACTIVE RHEUMATIC CARDIAC LESIONS IN PATIENTS SUBMITTED TO MITRAL COMMISSUROTOMY AND THE EFFECT OF CORTISONE ON THESE LESIONS

Clinical and Histopathologic Study of Sixty Cases

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IT HAS been several years since the first cases of mitral commissurotomy were reported.[1-5] During this lapse great experience has been accumulated.[6-11]

Through the analysis and detailed study of several hundred patients with mitral commissurotomy, the clinicians and surgeons have been able to know better the anatomic and physiopathologic aspects of this process, as well as the indications, contraindications, surgical techniques, risks involved, and results to be expected. However, an important problem remains unsolved; this is the one concerning the relatively high percentage of asymptomatic cases, (50 per cent or more according to various investigators) in which biopsy studies show active rheumatic lesions in the left atrial appendages at the time of commissurotomy.[12-16]

One of the aims of this paper is to point out the incidence of active rheumatic lesions in asymptomatic cases submitted to mitral commissurotomy, and whether there exist clinical or laboratory clues which might assist in making this diagnosis beforehand. We also observed the postoperative course, trying to obtain possible data which would allow the preoperative evaluation of the surgical risk.

In a group of patients, cortisone was given preoperatively in order to observe its effects on active but asymptomatic rheumatic heart cases, as well as upon postoperative relapses; in other words, to find out if ACTH or cortisone decreases or eliminates tissue inflammatory reactions of the heart, due to rheumatic activity as well as to surgical trauma. This is very important because the fibroblastic process of cicatrization can, with time, lead to a recurrence of valvular stenosis or other complications.

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MATERIAL AND METHODS

Between 1952 and 1953, a series of sixty patients to be submitted to mitral commissurotomy was studied. Fourteen nonselected cases were treated with cortisone, and the remaining forty-six patients served as a control.

All of them were carefully studied, searching for rheumatic activity and the degree of cardiac lesions. Complementary studies were done including blood chemistry, antistreptolysins, sedimentation rate, x-rays, phonocardiograms, and electrocardiograms. The group of fourteen patients treated with cortisone was divided into (1) those in whom a total daily dose of 100 to 200 mg. was given during a period of 4 to 10 days before surgical intervention; and, (2) a second group of patients who received cortisone from 1 to 3 months before surgery. The initial dose was the same as for the first group, and after 2 to 3 weeks it was reduced to 75 to 100 mg. daily up to the fifteenth postoperative day.

During the operation a biopsy specimen was taken from the left atrial appendage in all the cases, and also from the pericardium in a few of them. Histopathologic studies were carried out in all the cases.

Three groups have been considered according to their activity: the first with active lesions; a second group with rheumatic lesions in apparent healing stage; and the third group with already healed lesions. Table I shows the criteria utilized for the qualification of rheumatic activity.

Special attention was given to the clinical methods in reference to the postoperative condition of the heart. This was done considering the rheumatic aspect as well as the surgical trauma.

TABLE I. BASIS AND CRITERIA TO QUALIFY THE DEGREE OF RHEUMATIC ACTIVITY IN PATIENTS SUBJECTED TO COMMISSUROTOMY

STAGE OF RHEUMATIC LESIONS	FIBRINOID NECROSIS	POLYMORPHONUCLEAR AND EOSINOPHILIC INFILTRATION	ASCHOFF NODULES	ASCHOFFOID NODULES AND LYMPHOHISTIOCYTIC INFILTRATION	LYMPHOCYTIC INFILTRATION	VASODILATATION	ENDOCARDIAL TUMEFACTION	PERIVASCULAR, INTERSTITIAL, PERICARDIAL AND ENDOCARDIAL FIBROSIS
Activity								
First group	+ +	+ + +	+ + +	+ +	+ +	+ + +	+ +	0
Second group	0	+	+ +	+ +	+ +	+ + +	+ +	0
Third group	0	0	+	+ +	+ +	+ +	+	0
Healing Stage ?	0	0	0	+	+ +	+	0	+ +
Healed Stage	0	0	0	0	0	0	0	+ + +

RESULTS

Of the sixty patients, thirty-six (60 per cent) revealed active endomyocarditis on the histopathologic study; nine (15 per cent) showed signs of being

Fig. 1.

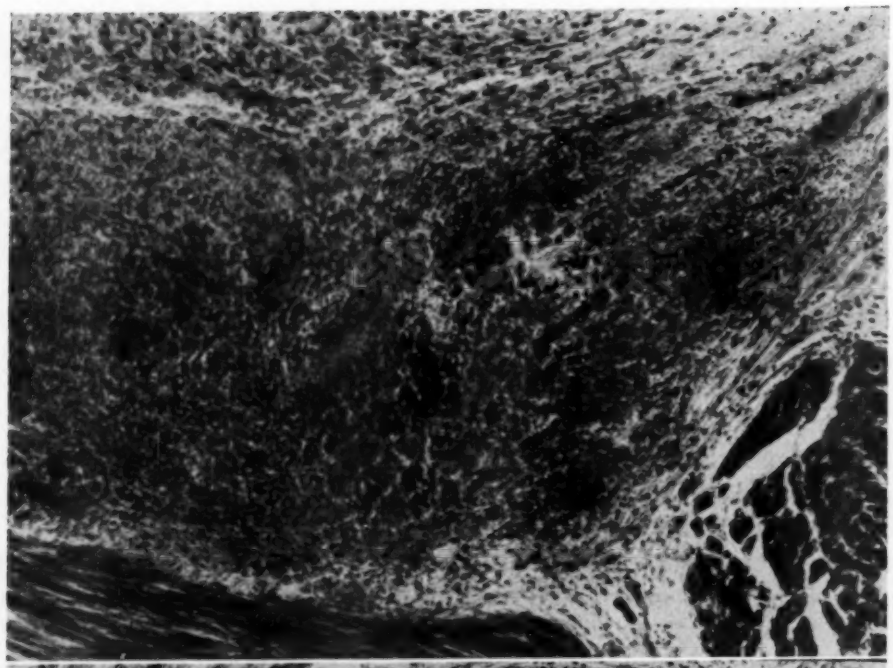

Fig. 2.

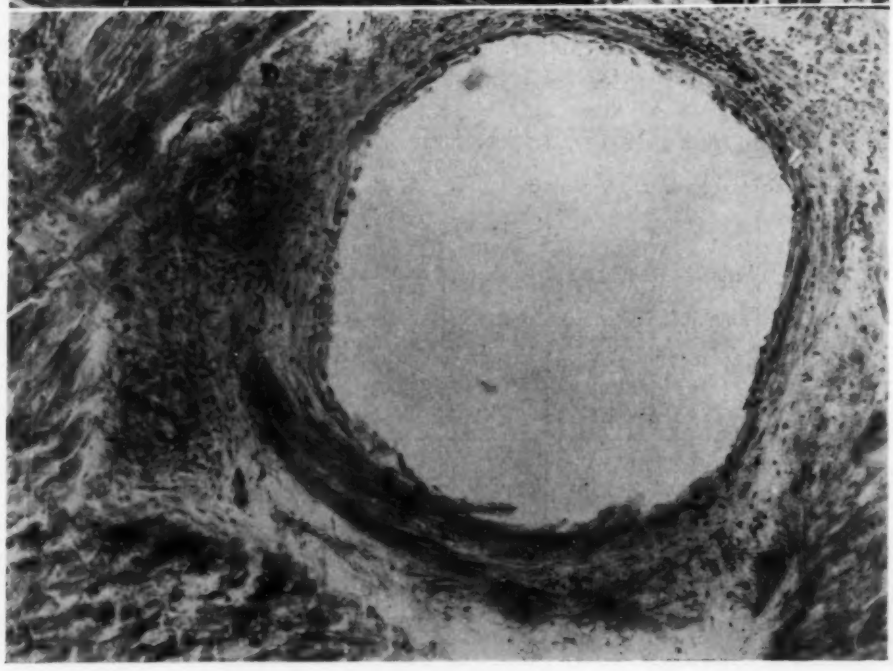

Fig. 1.—Histopathologic study showing numerous Aschoff nodules and severe leukocytic infiltration.

Fig. 2.—Section of auricular appendage showing less severe rheumatic activity with numerous perivascular Aschoff nodules.

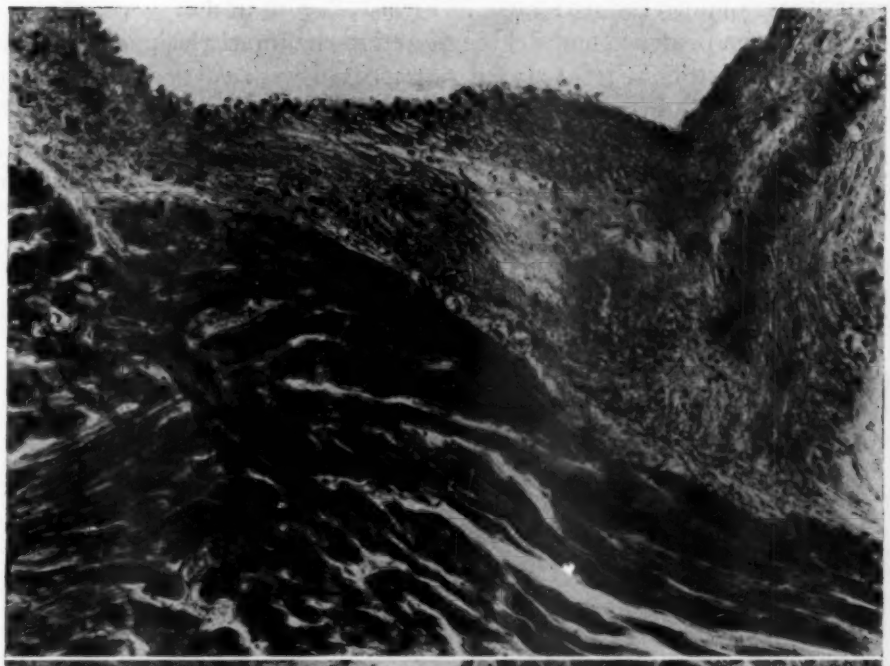

Fig. 3.

Fig. 4.

Fig. 3.—Endocardial Aschoff nodules.

Fig. 4.—Doubtful rheumatic activity in the endocardium without Aschoff nodules and slight lympho-histiocytic infiltration (healing rheumatic heart lesions?).

in the process of cicatrization; and fifteen (25 per cent) revealed healed lesions. The different stages of activity are shown in Figs. 1, 2, 3, 4, and 5.

If we analyze the presence and degree of active rheumatic lesions in relation to the proportion of patients treated and not treated with cortisone, a striking difference between both groups is found. Active rheumatic lesions are infrequent and less severe when cortisone is used. The ratio of treated to nontreated patients is 1/4.28, and, on the other hand, this proportion diminishes to 1/7.2 when the degree of rheumatic activity is taken into consideration. The opposite occurs when we analyze the relation between the two groups (Fig. 6), one in the process of healing and the other with healed lesions. The proportion here is 1/2.22 and 1/3, respectively.

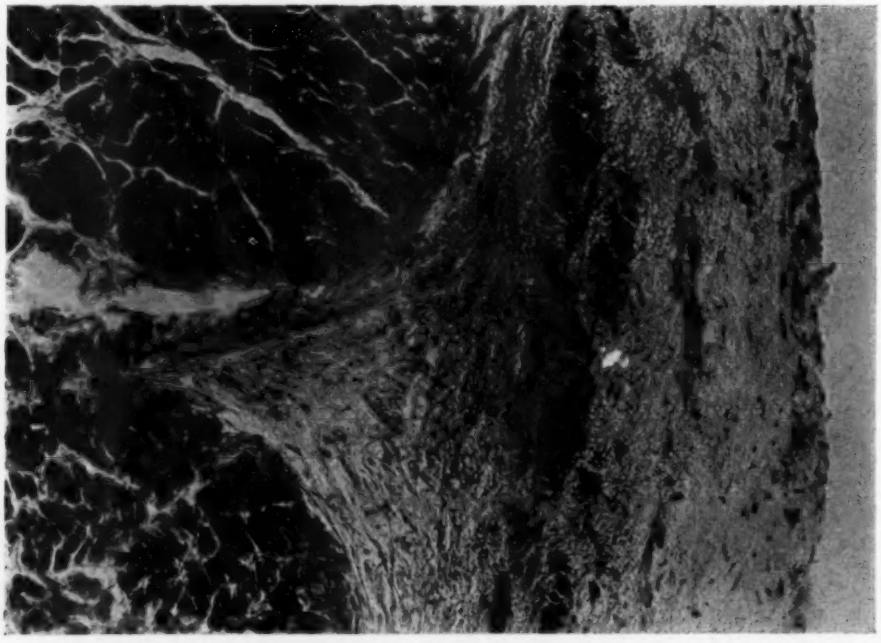

Fig. 5.—Healed rheumatic lesions of the endocardium with fibrosis.

In order to demonstrate this idea more clearly, it is necessary to study separately for the two groups, the percentage of patients with active and inactive lesions. Fig. 7 shows these percentages as sectors of circles and are (*A*) in the nontreated group, 67.3 per cent for the active lesions, 10.86 per cent for the lesions in the process of healing, and 21.7 per cent for those already healed; in circle (*B*) is shown the group receiving cortisone and the percentages are 35.7 per cent, 28.5 per cent, and 35.7 per cent, respectively. It is readily seen that the group receiving cortisone presents fewer active lesions.

Furthermore, none of the patients treated with cortisone belonged to the subgroup of greatest rheumatic activity. In the second subgroup there were four treated cases and three of them received the smallest dosage of cortisone.

Aschoff nodules did not disappear when cortisone was given for a short time, (4 to 10 days before operation) and at small dosage; even though other

signs of rheumatic activity diminished or disappeared. For this reason a more intensive and prolonged treatment (1 to 3 months) was given to three patients. Of these three cases, two presented entirely healed lesions, and the third case showed alterations suggestive of activity but without typical Aschoff bodies.

No relationship was found between past history of rheumatic activity and the occurrence of asymptomatic but active rheumatic lesions found on the histopathologic study.

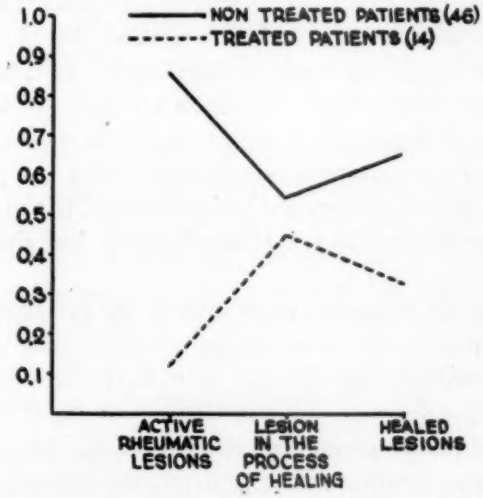

Fig. 6.—Ratio of the number of cases treated with cortisone as compared to those without cortisone (expressed in tenths of the total number of each group) and incidence of active rheumatic heart lesions.

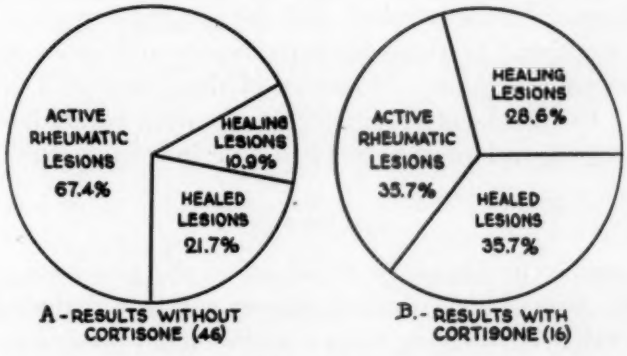

Fig. 7.—Comparative study of the incidence of cardiac rheumatic lesions in different stages of activity in patients treated with cortisone and in those without cortisone.

The number of postoperative relapses in the control group was 10.8 per cent as compared to 7.1 per cent of the group treated with the hormone. Relapse occurred several weeks or months after operation, except in two cases, in which it occurred immediately. These cases were from the untreated group.

From the clinical and phonocardiographic points of view, the postoperative pericarditis was less frequent and less severe in treated patients (38.4 per cent) than in the untreated group (86.3 per cent). The ECG studies demonstrated

similar results not only in regard to the pericardium, but also in respect to the myocardium. It was observed that the percentage of alterations compatible with a pericarditis in the treated group was 36.3 per cent, and 72.7 per cent in the untreated. From the histopathologic point of view, it was found that 21.4 per cent of the cases in the first group and 50 per cent in the second group showed these changes.

Almost 100 per cent of the operated patients, presented a leukocytosis count of 10 to 20,000 on the first postoperative day, and it generally disappeared in 3 to 10 days. The sedimentation rate was also found accelerated from 1 to 2-plus (tabulated from 1-plus to 3-plus according to its intensity). It appeared and disappeared more slowly than the leukocytosis, generally lasting several weeks. The degree and duration of leukocytosis as well as the velocity of the sedimentation rate had no apparent relationship with the presence and severity of asymptomatic but active rheumatic lesions found through biopsy. Perhaps these changes were less marked in the treated group, but the difference was slight and inconsistent.

The following ECG changes were noted in relation to mitral commissurotomy: (1) diminution of overloading (strain) and hypertrophy of the right ventricle (a decrease in the voltage of R in V_1 and of S in V_6; rotation of $\hat{A}_{QRS}$ to the left); (2) a decrease in the degree of right bundle branch block; (3) a small decrease in the notching and slurring of P, and at times of its duration and height; (4) the appearance of signs of strain and hypertrophy of the left ventricle, and, in some cases, of an increase in left bundle branch block.

It is very important that these results should be analyzed, since, in some cases, they could give rise to new complications even in spite of the improvement in the pulmonary hypertension, and the general hemodynamics.

The use of cortisone in these patients before and after operation did not cause any serious complication. However, of those treated 2 or 3 months prior to surgery, and 2 to 3 weeks after surgery, one patient had a delay in the healing of the surgical wound, and another one had a peripheral vascular collapse during operation.

DISCUSSION

It is not possible to discuss in detail all of the problems presented in this field of research. Only a few points of interest will be mentioned.

Keeping in mind the limiting nature of the biopsy study, one can appreciate how difficult it is to indicate exactly the incidence of asymptomatic but active rheumatic cases submitted to commissurotomy. Nor is it possible to arrive at categorical conclusions concerning the effect of cortisone on these lesions. However, it is evident that rheumatic activity occurs frequently in asymptomatic patients. It is also certain that cortisone has a beneficial effect on rheumatic active lesions as well as on those of traumatic nature due to surgery. This is shown not only by the histopathologic studies, but also by the clinical, electrocardiographic, and phonocardiographic findings.

Even though it was not possible to prove that the postoperative course was better in the group of patients treated with cortisone, we believe further studies are necessary. We feel that the lack of proof is not due so much to the

absence of results, but to the difficulty in evaluating them and to the nature of the process which merits a longer period of observation. The high incidence of rheumatic relapses in patients submitted to commissurotomy (10 to 30 per cent), as well as the appearance of the postcommissurotomy syndrome, which is considered as a special type of rheumatic activity by some authors, makes it necessary to establish a prophylactic treatment.

The use of cortisone or ACTH seems to aid in preventing such rheumatic relapses. In the group treated with cortisone none of the patients presented the postcommissurotomy syndrome, as compared with two instances in the untreated group.

SUMMARY

Sixty patients submitted to mitral commissurotomy were studied. Fourteen of them were treated with cortisone before as well as after surgery in order to evaluate (a) the incidence of asymptomatic but active cardiac rheumatic lesions; (b) the effects of cortisone on these lesions as well as upon the inflammatory reactions secondary to the surgical trauma, and (c) whether or not the postoperative follow-up in the treated patients is more favorable than in the untreated group.

CONCLUSIONS

1. A large group (60 per cent) of patients submitted to commissurotomy show active but asymptomatic rheumatic cardiac lesions.

2. The use of cortisone diminishes the presence of such lesions from 67.4 per cent to 35.7 per cent. It increases the frequency of lesions moving toward cicatrization or already healed from 10.9 per cent to 28.6 per cent and from 21.7 per cent to 35.7 per cent, respectively.

3. Cortisone can decrease the inflammatory surgical reactions of the pericardium and endomyocardium. Through clinical, phonocardiographic, electrocardiographic, and histopathologic studies, the frequency of pericarditis has been shown to decrease from 86.3 per cent to 38.4 per cent, from 72.7 per cent to 36.3 per cent, and from 50 per cent to 21.4 per cent, respectively.

4. It was not possible to clearly establish a better evolution in the postoperative period in those patients treated with cortisone as compared with the untreated. However, relapses and postcommissurotomy syndrome were less frequently observed in the first group. The results show that further investigations are needed.

SUMMARIO E CONCLUSIONES IN INTERLINGUA

Ex un gruppo de 60 patientes subjicite a commissurotomia mitral, 14 recipeva cortisona tanto pre- como etiam post-operativemente.

Lesiones cardiac rheumatic del typo active sed asymptomatic esseva constatate in 60 pro cento del gruppo integre. Le uso de cortisona diminueva le presentia de tal lesiones ab 67,4 a 35,7 pro cento; illo augmentava le procentage de lesiones in processo de cicatrisation ab 10,9 a 28,6 e le procentage de lesiones jam cicatrisate ab 21,7 a 35,7. Le uso de cortisona diminueva etiam le frequentia

de pericarditis postoperative. Observationes clinic e phonocardiographic revelava un diminution de iste condition ab 86,3 a 38,4 pro cento. Secundo observationes electrocardiographic le diminution esseva ab 72,7 a 36,3 pro cento, e secundo observationes histopathologic illo esseva ab 50 a 21,4 pro cento.

Relapsos e syndrome postcommissurotomic esseva minus frequente inter patientes tractate con cortisona que inter le alteres. Nostre resultatos indica que investigationes additional es requirite.

The cortisone used in this research was available through the courtesy of Merck & Co.

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PECTUS EXCAVATUM ("FUNNEL BREAST"), A CAUSE OF IMPAIRED VENTRICULAR DISTENSIBILITY AS EXHIBITED BY RIGHT VENTRICULAR PRESSURE PATTERN

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SINCE the work of Hansen and his associates,[1] the recognition of a pattern of the right ventricle pressure in conditions impairing the ventricular distensibility has been described in a multitude of abnormal states arising from the heart structure or its serous covering.[2-6] Another factor which has hitherto been undescribed is reported in this paper. The impairment of the ventricular filling is due to a pectus excavatum ("funnel breast"). This condition in the patient studied produces a typical pattern of a diastolic dip and plateau in the pressure curve of the right ventricle. Fig. 1 is an illustration of the right ventricular pressure as recorded during catheterization of the right heart. Table I summarizes the recordings of the pressure.

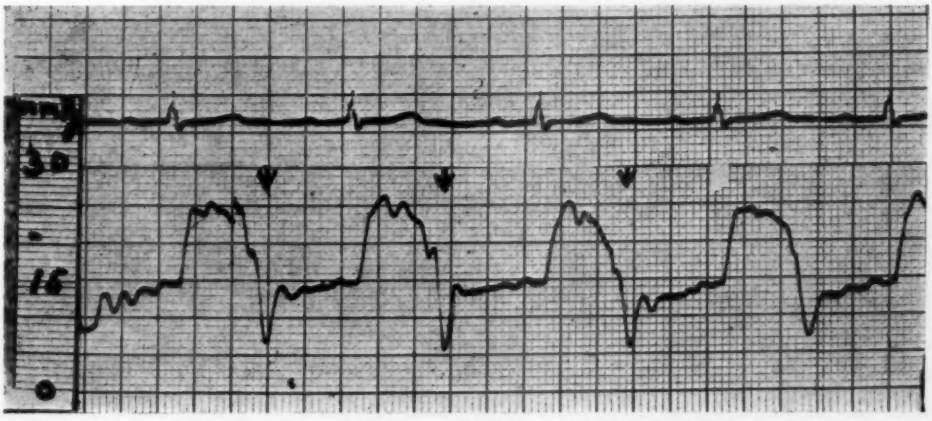

Fig. 1.—Simultaneous electrocardiogram and right ventricular pressure recordings during right-heart catheterization. The arrows point to the diastolic dip which is followed by the plateau at a high level of pressure

The systolic levels of pressure in the right ventricle are normal except for the end diastolic pressure which is elevated. The normal value is 0 to 5 mm. Hg, but in this patient it is recorded at 11.5 to 12.5 mm. Hg. The right auricular mean pressure is likewise elevated to the same degree. The contour of the right

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ventricular curve is of the type that is found in constrictive pericarditis in which there is a diastolic dip and a plateau. The plateau is at a high level of pressure at the end of diastole, indicating a compressive effect on this ventricular chamber. The skeletal defect produces this pressure abnormality in the right ventricle. The normal wedge pressure of the pulmonary artery indicates that the left ventricle is not embarrassed by the skeletal deformity.

TABLE I. PRESSURE READINGS IN MM. HG RECORDED IN A CASE OF PECTUS EXCAVATUM DURING RIGHT-HEART CATHETERIZATION

LOCATION OF TIP OF CATHETER	PRESSURE IN MM. HG		
	SYSTOLIC	DIASTOLIC	MEAN
Pulmonary capillary			7–10
Pulmonary artery	23–25	11.5–12.5	17.5
Right ventricle	23–26	11.5–12–5	
Right auricle			12.5

SUMMARY

An extracardiac and extrapericardial cause for the typical pattern of right ventricular pressure, usually described as due to constrictive pericarditis, is reported.

SUMMARIO IN INTERLINGUA

Es reportate un causa extracardiac e extrapericardiac pro le typic configuration del pression dexteroventricular que es usualmente describite como resultante de pericarditis constrictive.

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A SIMPLE METHOD FOR OBTAINING ESOPHAGEAL ELECTROCARDIOGRAMS OF GOOD DIAGNOSTIC QUALITY

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ESOPHAGEAL electrocardiograms may be of considerable aid in the study of atrial abnormalities, particularly arrhythmias,[1] since they provide records of atrial activity which closely resemble direct leads from this region.[2] They are also of some value in the study of ventricular abnormalities because they provide semidirect leads from the inferoposterior aspect of the left ventricle[2,3] which in conventional electrocardiography may be a "blind" area. From higher levels in the esophagus the recorded ventricular complexes resemble intracavitary potentials.[2]

Despite these meritorious features of esophageal leads it is our impression that they are not widely employed in clinical practice because of technical difficulties, particularly excessive wandering of the base line of the tracings. On the supposition that this base-line instability is largely due to variable contact potential at the metal tip of the conventional esophageal electrode, we employed as an esophageal electrode a simple electrolyte bridge consisting of 0.85 per cent sodium chloride solution within a plastic stomach tube.* We have observed that esophageal electrocardiograms obtained in this manner exhibit relatively stable base lines.

The tube is modified from its original form by cutting off the terminal portion which contains side fenestrations. The cut end is softened by several minutes of immersion in acetone, and then rounded by pressing it within a hot, tapered cylindrical mold. The glass portion of an ordinary medicine dropper serves this purpose well. This procedure narrows the terminal portion of the tube lumen to approximately a 1 mm. diameter. Any residual roughness of the rounded tip may be removed by rubbing with fine sandpaper. The tube assembly is completed by forcing the hose end of a metallic adapter† into the proximal end of the tube. The male end of a metallic stopcock with hypodermic syringe and needle fittings‡ is inserted into the open end of the adapter. The electrical resistance of the simple saline column is so large (75,000 ohms)

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*K-10 expendable Kaslow stomach tube (Levine type), 16 French, Pharmaceal Laboratories, Glendale 1, Calif.

†L/606 Adapter, Becton, Dickinson and Company, Rutherford, New Jersey.

‡L/84 One-way valve stopcock, Becton, Dickinson and Company, Rutherford, New Jersey.

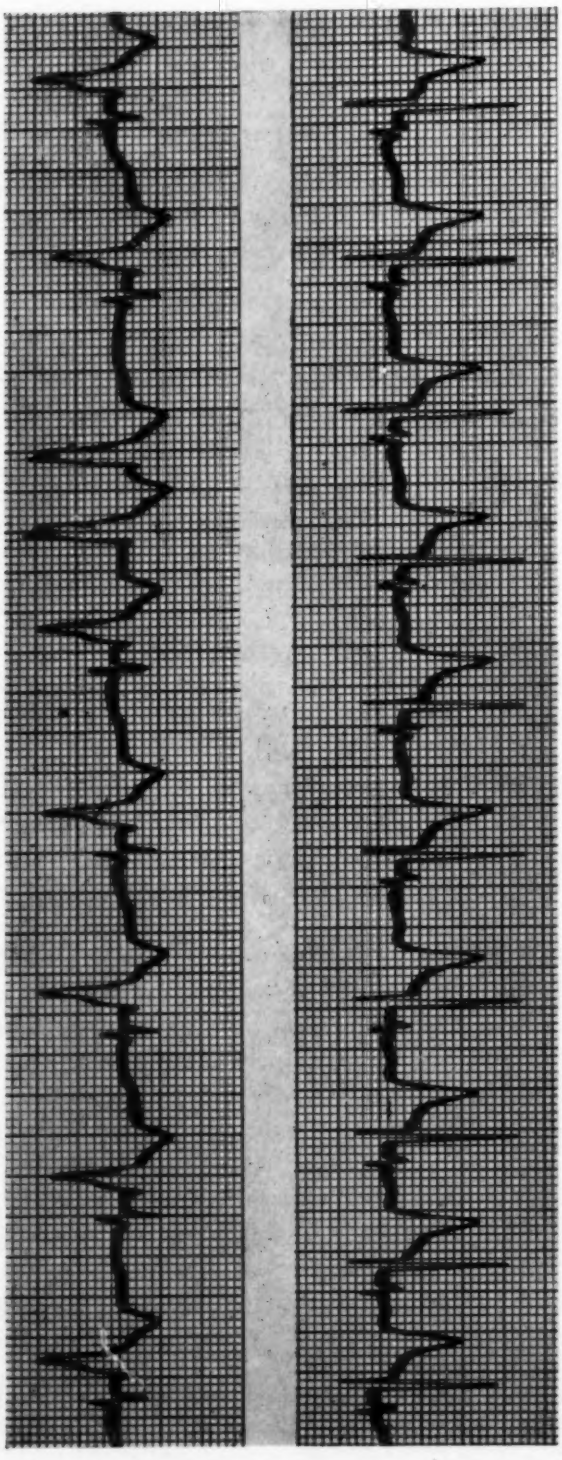

Fig. 1.—Specimen tracings of esophageal leads taken at atrial transition levels by means of the technique described in the text. Upper strip: 70-year-old white male with hypertensive cardiovascular disease, left bundle branch block, and premature ventricular contractions. Lower strip: 19-year-old Negro woman with hypertension due to acute glomerulonephritis.

that there is some loss of magnitude in the registration of intraesophageal potentials, and the tracings frequently exhibit too much AC interference. These objections are overcome by introducing No. 31 gauge stainless steel suture wire throughout the length of the saline column. The distal end of the wire is rolled into a ball for the purpose of reducing contact resistance and preventing the wire from passing beyond the tip of the tube lumen. The proximal end of the wire is threaded into the lumen of the tube adapter and through a small hole drilled in the side of the adapter. It is then wound around the neck of the adapter and silver-soldered in place. The resistance of this assembly, as measured by a 1 kc./sec. impedance bridge, is 1,000 to 5,000 ohms depending largely on the distance of the end of the steel wire from the distal end of the tube. After lubrication of its distal end with mineral oil, the tube is passed pernasally until it reaches the pharynx. The patient is then given a glass of water to drink and, while he is swallowing, the tube is easily and rapidly advanced to a distance which insures entrance of its tip into the stomach. After the tube has been filled with normal saline solution, the stopcock is connected to the appropriate input lead of an electronic electrocardiograph. As the tube is retracted in 2 cm. intervals, an electrocardiogram is recorded at each step of withdrawal. Under these conditions the base line of the records exhibits satisfactory stability except when the tip is in the region of the cardiac sphincter. In some subjects there is undulation of the base line in synchronism with respiratory movements. This undesirable feature is eliminated and a satisfactory record obtained by having the subject suspend respirations for several seconds. The erratic, widely swinging base line deviations which are commonly associated with the use of a German silver esophageal electrode rarely occur and are greatly minimized with the salt-bridge technique. Occasionally considerable AC interference develops during the procedure. This is apparently due to the entrance of a bubble of gas into the distal end of the fluid column and may be corrected by flushing the tube with additional saline solution.

Probably there are fewer contraindications to the use of this esophageal electrode than there are to the use of the more conventional type. The tubing is so pliable and smooth that our subjects appear to suffer very little distress during the introduction of the tube, and virtually no discomfort during the remainder of the procedure. For this reason we have felt justified in recording esophageal electrocardiograms from subjects in whom there was a question of recent posterior myocardial infarction. We have also successfully passed the electrode in an unconscious patient who had a diagnostically difficult arrhythmia.

The salt bridge is commonly employed in electrophysiologic techniques, and it has been successfully applied to the registration of cardiac intracavitary potentials.[4,5] However, a careful search of the literature available to us has failed to reveal that it has been used for the registration of esophageal electrocardiograms. Apparently this application of the salt-bridge technique has either not been previously described or else it is not widely known.

The electrode assembly described here may be easily and economically constructed from standard components available at surgical supply houses. Construction may be simplified by not soldering the proximal end of the wire

to the neck of the adapter since the plastic tubing can be employed to hold the wire in place and to occlude the side hole which was drilled in the adapter. Because of the simplicity and reliability of the method of esophageal electrocardiography reported here, we believe that it should be called to the attention of those who may be interested in this field of study.

SUMMARIO IN INTERLINGUA

Le curvate deviationes erratic del linea de base que es normalmente characteristic de electrocardiogrammas esophagee obtenite per medio de electrodos conventional a puncta metallic, es apparentemente causate per un variabile potential de contacto al extremitate distal del electrodo. Iste defecto es practicamente eliminate per le uso de un simple ponte electrolytic que consiste in principio de un solution salin physiologic intra un plastic sonda gastric. A causa del simplicitate e efficacia de iste methodo illo es portate al attention del interessatos in le campo de electrocardiographia esophagee.

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Clinical Reports

HALVING OF THE PULSE DUE TO SEVERE ALTERNANS (PULSUS BISECTUS)

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CASE REPORT

History.—This 46-year-old Negro was sent to the Cardiac Clinic after hospitalization at Mount Sinai Hospital from Jan. 3, 1953 to Feb. 1, 1954. The patient complained of exertional dyspnea, orthopnea, cough, and swelling of the abdomen for 2 years. There had been edema of the ankles for several months. Recently, several attacks of nocturnal dyspnea occurred. The patient improved after bed rest, low-salt diet, Mercuhydrin, and digitoxin. The patient stated that he had rheumatic fever when he was about 10 years old, with swelling of several joints and fever. In 1951, he was told by a physician that he had high blood pressure. Since then, he has been treated with digitalis and Mercuhydrin and placed on a low-salt diet.

Physical Examination.—The patient is a husky man in no acute distress. Moderate dyspnea upon exertion. No cyanosis. Blood pressure 110-122/86-90 mm. Hg. Pulse 84/min. and regular. The jugular veins are not distended in a sitting position.

Lungs: Clear.

Heart: Visible and diffuse apical impulse in the fifth and sixth intercostal spaces 2 cm. beyond the midclavicular line. The first sound at the apex is weak and is followed by a Grade 2, soft, systolic murmur. This murmur is transmitted to the midprecordium. P_2 is slightly louder than A_2. A_2 at times seems to be alternating in intensity.

The *liver* edge is palpable two fingerbreadths below the right costal border and is not tender. There is a 1+ edema of the ankles.

Laboratory Findings.—

Blood: Red blood count, 4.3 million; hemoglobin, 12.8 grams; white blood count, 9,850; differential count, normal. Total serum protein = 7.7; A/G ratio = 1.4; serum electrolytes within normal limits.

Urine: There was a variable amount of albumin in the first three weeks of hospitalization. Since then, the urine has become free of albumin. Spec. grav. = 1.010 – 1.026. No sugar; no blood; no casts.

X-ray.—The chest film revealed a severely enlarged heart. Enlargement of the left atrium and ventricle was noted in left anterior oblique (LAO); enlargement of the right atrium and ventricle was noted in right anterior oblique (RAO).

Electrocardiogram.—Sinus rhythm with a regular rate of 60/min. No electric alternans. First degree A-V block. Evidence of left ventricular hypertrophy. Horizontal heart. Antero-lateral ischemia (Figs. 1 and 2).

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Phonocardiogram.—Faint systolic murmur at the apex. The first sound was of low amplitude; the second sound was split over the pulmonic area. In some of the tracings, alternation of the heart sounds was noted: when the pulse was larger, the second sound was louder (Fig. 3).

Pulse Tracings.—Pulse tracings were recorded at the arm through a pneumatic cuff with variable compression, and over the carotid artery. In most tracings, severe alternation was noted (Fig. 4,*A*). In some of them, a complete disappearance of the small waves occurred, even when the compression exerted on the artery was extremely slight (Fig. 4,*B*). When a common type of alternation took place, the small pulse waves were nearer to the following than to the preceding waves (distances: 0.15–0.18–0.15–0.18). The position of the patient had some influence on the pulse. Alternation rapidly disappeared as soon as the patient was placed in the supine position, but it frequently reappeared after a deep breath.

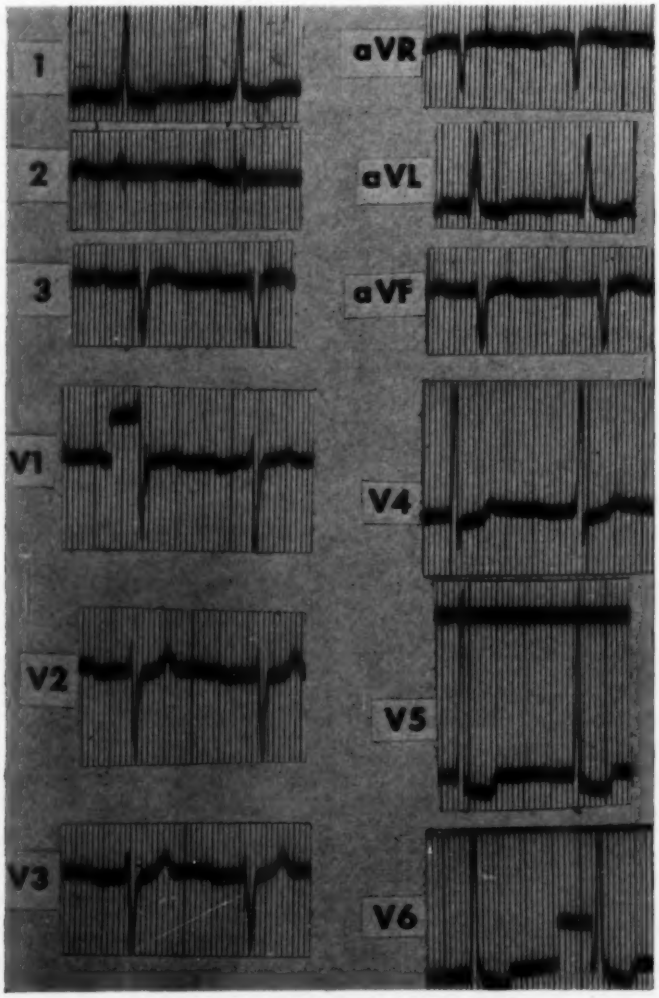

Fig. 1.—Electrocardiogram. No alternans.

Electrokymograms.—The tracings of the left ventricular border revealed a severe alternation over the entire contour, both in the posteroanterior and in the oblique positions. The small pulsations were preceded by an incomplete diastole (Fig. 5,*A*). The tracings of the right atrium showed no alternation. Those of the left atrium showed a definite alternation, but the variation occurred mostly during ventricular contraction. The tracings of the pulmonary knob revealed a severe alternation (Fig. 5,*B*). The tracings of the aortic knob, on the other hand, revealed at times the complete absence of one pulsation out of two and halving of the pulse (Fig. 5,*C*).

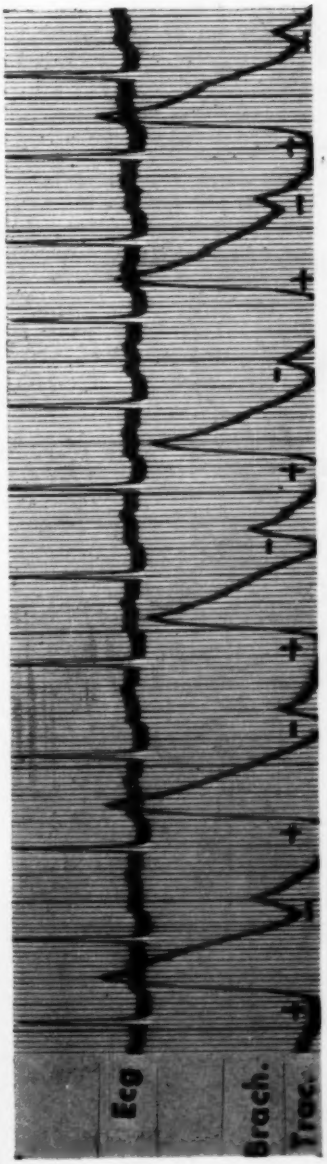

Fig. 2.—Electrocardiogram and brachial tracing. Pulsus alternans. The tracing was recorded at a cuff pressure of 90, equivalent to diastolic pressure.

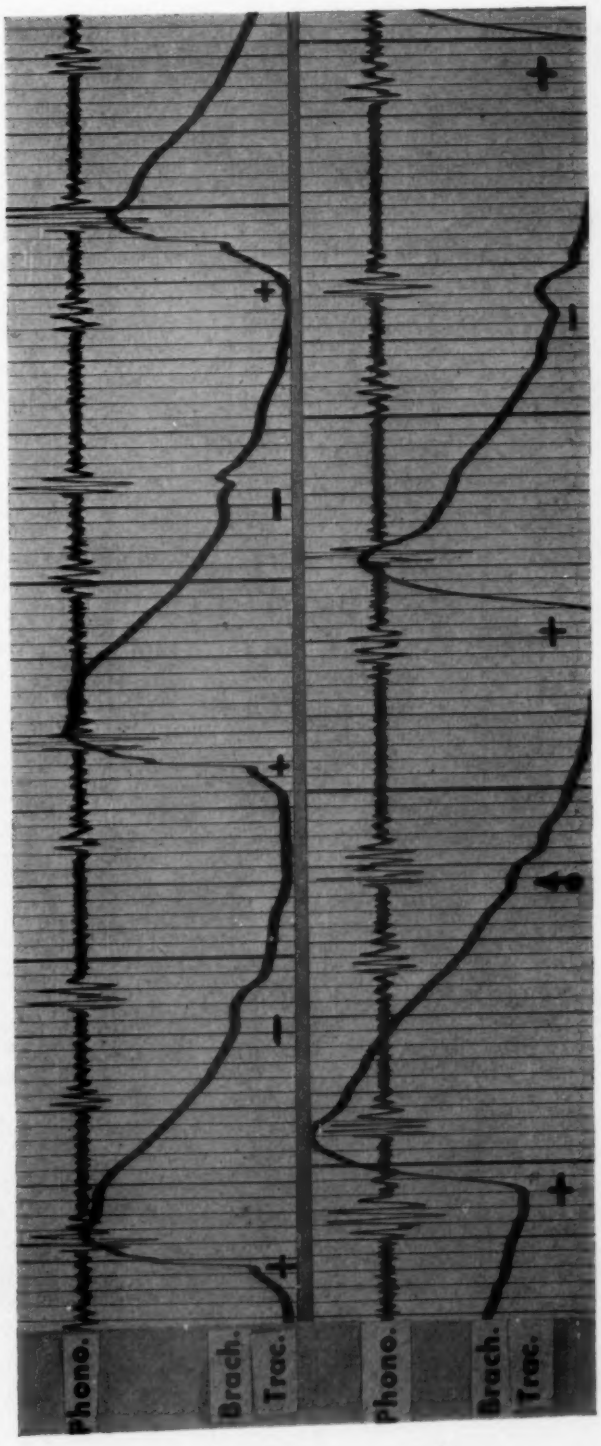

Fig. 3.—Phonocardiogram and brachial tracing at 90 mm. Alternation of heart sounds (discordant alternation of the first sound; concordant alternation of the second sound). Small pulse waves are visible in cycles 2 and 4 of tracing *A*, and in cycle 6 in tracing *B*; there is a questionable pulse wave in cycle 2 of tracing *B*.

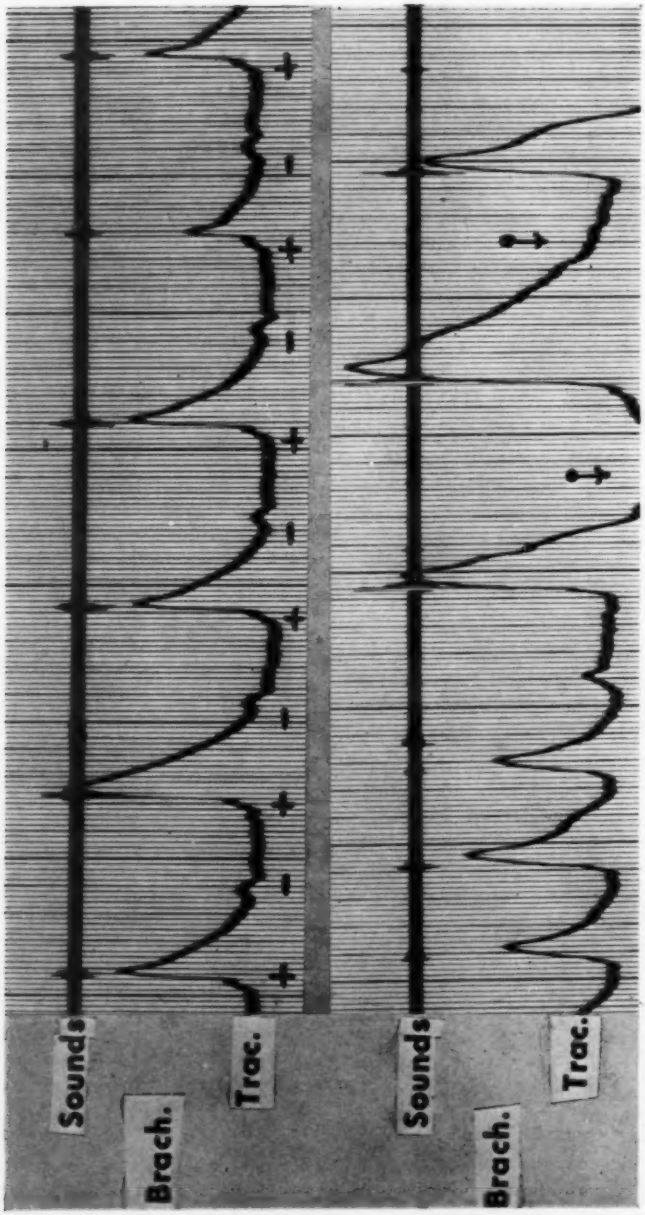

Fig. 4.—Brachial tracings: above = arterial sounds; below = arterial pulses. Both are simultaneously recorded by two crystal microphones from a recording cuff inflated at 25 mm. The compressing cuff had a pressure of 90, equivalent to diastolic pressure. Cycles 2, 4, 6, 8, and 10 of tracing *A* have an extremely small pulse wave but no arterial sound (stethoscopic halving). In tracing *B*, a deep respiration disturbs the sequence causing larger pulses: cycles 6 and 8 have no pulse and no arterial sound (sphygmographic and stethoscopic halving).

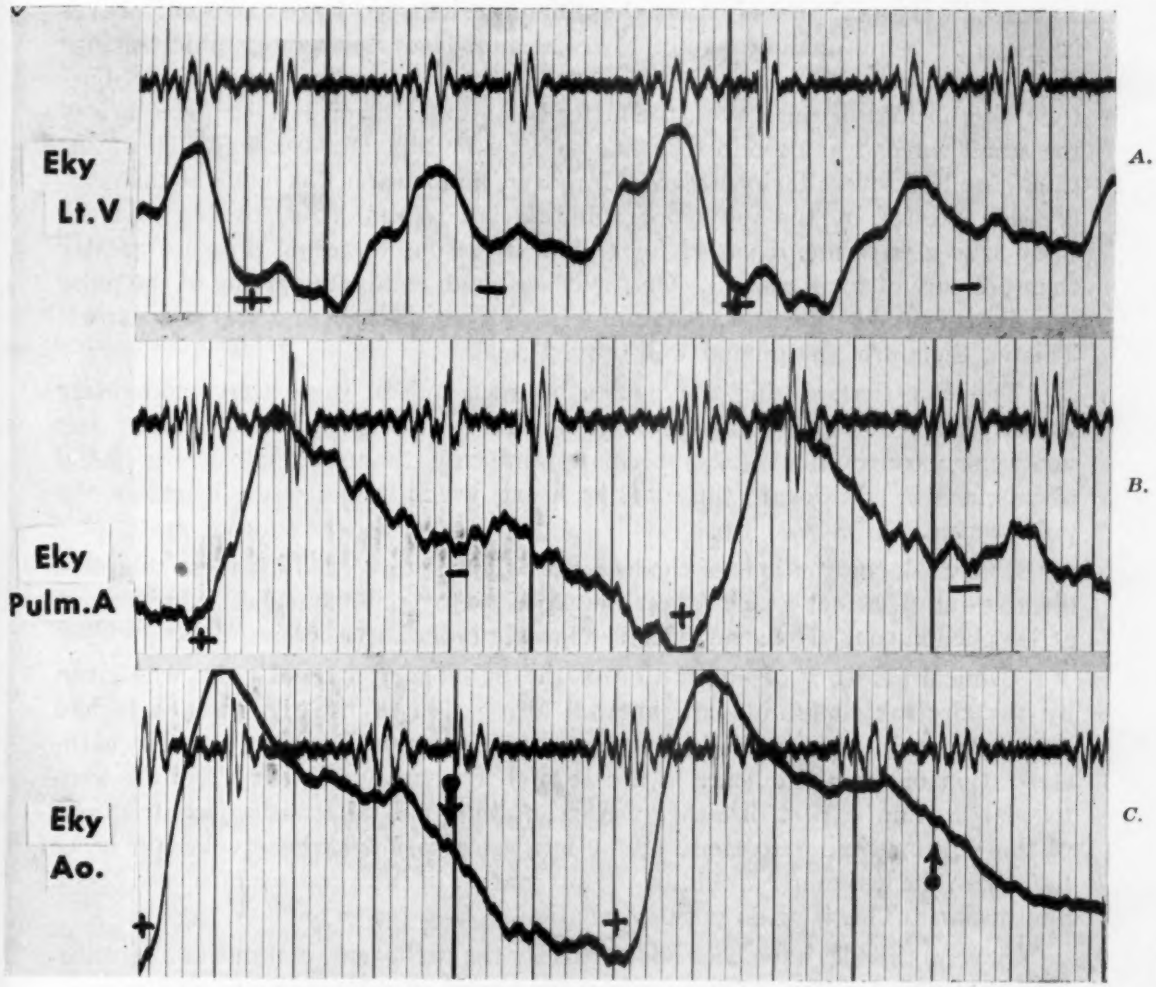

Fig. 5.—Electrokymograms and simultaneous sound tracings. *A*, *Eky* of left ventricular border in PA. Alternation of diastole and systole with smaller waves in cycles 2 and 4. *B*, *Eky* of pulmonary knob. Alternation of pulmonic pulses. *C*, *Eky* of aortic knob. Complete halving of the pulse. The small waves of cycles 2 and 4 should appear at the time of the second sounds (arrows).

DISCUSSION

Alternation of the pulse was well known at the end of the last century. Alternation of the heart, while documented early in animal experimentation, was observed only much later in clinical cases. Alternating intensity of the heart sounds or murmurs,[1,3-8] alternation of the force of the apex beat,[8] and alternation in the height of the ventricular contractions recorded by either the roentgenkymograph,[9,10] or the electrokymograph[11,20,21] have been described in the last 20 years.

In general, pulsus alternans is detected either by palpating the radial artery or during blood pressure measurement. The latter, not only reveals the phenomenon, but also indicates the pressure levels of both the large and the small

waves. In the majority of cases, the difference between large and small waves is moderate being in the range of 15 to 20 mm. Hg. Sphygmographic tracings may reveal alternation of either the peak (systolic alternation) or the foot (diastolic alternation) of the waves, the latter being much rarer.[12] Disappearance of the small waves was described in one case by Padilla and Cossio.[19] However, their case had severe tachycardia. Moreover, no mention was made of the compression exerted by the sphygmographic device, so that it is possible that the small waves were not recorded for technical reasons (external pressure greater than pressure of small pulses). On the other hand, complete halving of the pulse was documented by electrokymography in a case of Blumberger and associates.[21] This is, therefore, the second well-proved case.

The fact that, during the pauses between pulses, there were moderately weaker heart sounds (phonocardiogram) and about 50 per cent weaker left ventricular contractions (eky) seems to indicate the existence of peripheral phenomena in addition to those of the heart, which modified the height of the pulse waves.

The weak contractions of the left ventricle were preceded by a less complete diastole (eky), a fact which might be explained by pulmonary vascular, or left atrial phenomena. However, this is a purely speculative assumption.

Clinical evidence of alternation of the pulmonary arterial pulse was given by the electrokymogram (experimental alternation of the right ventricle had been previously described[18]) in a case of Blumberger and associates[21] and by catheterization of the right heart in a case of Katz and associates.[22] As the ventricular septum is part of either ventricle, abnormal (alternating) contractions of the septum similar to those of the left ventricle might be invoked in order to explain the less marked pulmonary alternation in the presence of severe aortic alternation. This is again pure speculation.

Several theories have been advocated for the pathogenesis of pulsus alternans. One of them admitted a longer refractory period in a limited area of the myocardium so that a section of the ventricle would contract only once every two stimuli ("partial asystole" of Gaskell[13] and Hering[14]). A step-by-step electrokymographic study of the left ventricle in our case failed to reveal either normal areas or more severely affected sections. This might be interpreted as indicating either wide distribution of fibers with a longer refractory period or diffuse alternation (global hyposystole of Wenckebach,[15] Frédéricq,[16] and Kisch[17]).

Wiggers' observation[2] that the larger beats are frequently larger than normal beats can be confirmed by this case. Whenever "halving" of the pulse started, there was an initial beat which was larger than the preceding ones (Fig. 4, *B*). The phenomenon was frequently started by a deeper respiration which probably modified the venous return. In most clinical cases, such a phenomenon starts pulsus alternans. In this case, where alternans already existed, a change of venous return caused "halving."

The influence of the standing or sitting position in favoring alternans was already noted by Friedman and associates.[20] It revealed the importance of dynamics in addition to myocardial factors in causing alternation.

SUMMARY

A striking case of pulsus alternans with alternating disappearance of the pulse is described. The names "halving of the pulse" or *pulsus bisectus* are suggested for this phenomenon. The electrokymographic tracings revealed alternans of the entire left ventricle and of the pulmonary pulse, and "halving" of the aortic pulse.

ADDENDUM

The patient was again examined one year after the first examination, and no alternans was found at this time.

SUMMARIO IN INTERLINGUA

Es describite un caso frappante de pulso alternante characterisate per un alternante disparition del pulso. Le terminos "bisection del pulso" o "pulso bisecte" es proponite pro iste phenomeno. Le electrokymogramma revelava "bisection" del pulso aortic e pulso alternante del integre ventriculo sinistre e del pulso pulmonar. Le patiente esseva re-examinate un anno post le prime examine e nulle pulso alternante esseva constatate.

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INTRACAVITARY CARDIAC MASSES: DIAGNOSIS AND TREATMENT

Report of Case of Ball Thrombus

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PATIENTS with cardiac myxomas or ball thrombi may be restored to health if the lesion is recognized. The present report is of a ball thrombus of the heart which could have been diagnosed during life, and with proper treatment the patient's life might have been saved.

CASE REPORT

A 57-year-old man was hospitalized because of marked dyspnea. Three years before he had been hospitalized because of severe dyspnea; he was discharged with the diagnosis of rheumatic heart disease and pulmonary emphysema. A year later he was again hospitalized because of dyspnea. Digitalization did not relieve his dyspnea.

On examination the patient was stuporous, severely dyspneic, and cyanotic. Respirations were of the Cheyne-Stokes type. The breath sounds were harsh and were associated with râles and rhonchi on the left side. The left border of cardiac dullness was at the anterior axillary line; the heart tones were barely audible; and extrasystoles were present. The liver edge was felt 4 cm. below the costal margin. The extremities were cold and cyanotic. Clubbing of the finger tips and moderate pitting edema of the lower extremities were present. On neurologic examination the head and eyes deviated to the left. The pupils were round, regular, and equal. There was sagging of the left corner of the mouth. Babinski's sign was present bilaterally, being more pronounced on the right. The left upper extremity was spastic, and the tendon reflexes were hyperactive.

On the seventh day of hospitalization, the patient experienced severe retrosternal pain. He died on the next day. An electrocardiogram prior to death showed complete atrioventricular dissociation and runs of ventricular tachycardia.

Autopsy revealed scattered petechiae throughout the white matter of the brain. The lungs were emphysematous and markedly congested and edematous. The right atrium and auricular appendage were hemorrhagic and contained yellowish mural thrombi. The right side of the heart was moderately dilated. Located within the right atrium was a red mass 5 by 5.5 by 8.5 cm., attached to the interatrial septum between the opening of the coronary sinus and a patent foramen ovale. It had a short pedicle 2.5 cm. in diameter. The mass was not laminated. The tricuspid valve was greatly dilated by the mass. There was no evidence of mitral stenosis. Microscopic examination showed the mass to be an organized ball thrombus.

CLASSIFICATION

Three types of masses may be found within the chambers of the heart. The first group consists of the free ball thrombus which, according to Welch,[1] must meet the following criteria: ". . . (1) entire absence of attachment and

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consequent free mobility; (2) imprisonment in consequence of excess in the diameter of the thrombus over that of the first narrowing in the circulatory passage ahead of it; and (3) such consistence and shape that the thrombus must not of necessity lodge as an embolus in this passage." The second group is composed of the pedunculated ball thrombi which differ from the first because of the endocardial attachment. The third group consists of the myxomas.

To avoid ambiguity of the term ball thrombus, Evans and Benson[2] suggested the term "mass thrombi" to include both free and pedunculated ball thrombus. We have chosen the clinical term "intracavitary cardiac masses" to include not only the free and pedunculated ball thrombi but also the myxomas.

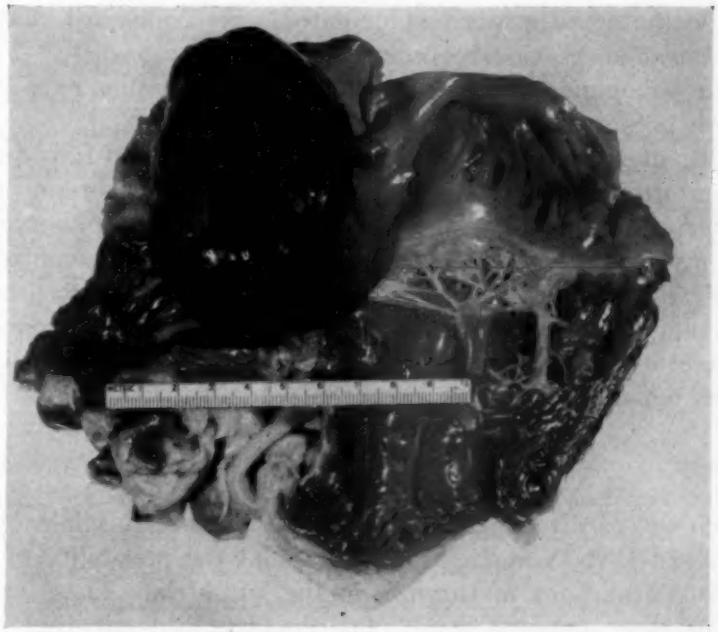

Fig. 1.—Ball thrombus in right atrium.

We have done this for the following reasons: (a) the three groups cannot be distinguished clinically with certainty; (b) the mechanism by which they produce symptoms is identical; and (c) the diagnosis and treatment of these three lesions are similar.

INCIDENCE

Wood[3] reported a case of ball thrombus in 1814. In 1950, Strade[4] stated that thirty-five cases of ball thrombi (including free, pedunculated, and large vegetative) had been reported. Goldberg and associates[5] in 1952, stated that approximately 350 cases of primary tumors of the heart have been reported of which approximately 126 were myxomas.

PATHOGENESIS

In 1890, Von Ziemssen[6] postulated that mitral stenosis was a necessary factor in the formation of ball thrombi. Although most cases have been associated with mitral stenosis, three cases mentioned by Strade,[4] in 1950, occurred in patients with hypertensive heart disease unassociated with mitral stenosis, but with atrial fibrillation. In a review of thirty-one cases of ball thrombi by Aronstein and Neuman,[7] in 1939, mitral stenosis was found in every case and it was severe in most cases. The observation was made that free ball thrombi were present only in cases with mitral stenosis while pedunculated ball thrombi have occurred in other conditions such as hypertension.

Free and pedunculated ball thrombi may have a common origin. Potter[8] reported a case with two ball thrombi in the left atrium. The larger thrombus was free while the smaller was pedunculated. He concluded that the older, larger thrombus had previously broken off the same pedicle. Fleming and Joels[9] had similar opinions and stated that a ball thrombus may break off its pedicle when it becomes large and heavy.

Even with material for microscopic examination, confusion may exist as to whether an intracavitary cardiac mass is of myxomatous origin or whether it is an organized thrombus. Ewing[10] was of the opinion that the vast majority of reported myxomas were genuine tumors rather than organized thrombi. Husten[11] critically reviewed seventy-one cases in 1923 and accepted only nine as true myxomas. Yater[12] believed these tumors to be of neoplastic origin. Although there are differences of opinion, Saphir[13] has described distinguishing characteristics between myxomas and organized ball thrombi.

DIAGNOSIS

Intracavitary cardiac masses have been diagnosed clinically, although infrequently. In 1890, Von Ziemssen[6] suggested the possibility of recognizing the clinical manifestations of thrombi in the left atria. These criteria based on a series of three patients were: (a) circumscribed gangrene of the feet; (b) cadaveric coldness and swelling of the feet; (c) absent or diminished arterial pulsations of the larger vessels of the lower extremities; and (d) high-grade mitral stenosis.

The pathognomonic signs and symptoms of intracavitary cardiac masses are based on intermittent obstruction of one of the cardiac orifices. This results, as quoted from Elson[14] in ". . . comparatively rapid and transitory changes in the peripheral circulation, such as marked cyanosis or even gangrene which may involve the finger tips, toes, or tip of the nose. Cadaveric coldness may occur suddenly, and quickly improve or disappear. The disappearance or diminution of pulsations, not from one extremity but from several of them, including both upper and lower, and their relatively rapid restoration . . . should be emphasized."

Because of its mobility, the intracavitary mass is influenced by gravity. Therefore, the symptoms and signs of cardiac obstruction may occur or disappear by changes in position. In the case reported by Houck and Bennett[15] the pa-

tient was a 44-year-old woman who had five fainting spells, all when she was in the upright position. Autopsy revealed a pedunculated left atrial mass. These authors felt that the fainting could have been caused by the tumor dropping into the mitral orifice and that the subsequent fall to the horizontal position may have dislodged it. Our patient had a distinct tendency to lie on his left side. This was so great that unilateral decubitus developed. The relief he gained apparently resulted from displacement of the atrial mass away from the tricuspid orifice by gravity. In contrast to these examples, the patient with heart disease is usually more comfortable in the upright position.

Coulter[16] stated that the diagnosis of a cardiac neoplasm should be entertained in cases of severe cardiac disease in which the signs cannot be correlated with the usual etiologic types of heart disease. This should be considered especially if the dyspnea is out of proportion to the physical findings. Schwartz and Biloon[17] emphasized the fact that they were unable to slow the ventricular rate with digitalis in three of their patients. They also mentioned the disappearance of the first heart sound.

As previously mentioned, most cases of intracavitary cardiac masses are associated with mitral stenosis. Likewise the majority of cases are associated with atrial fibrillation. The intracavitary mass is usually located in the left atrium.[7] The diagnosis should be entertained in every case of marked mitral stenosis of long duration as it may be associated with or be mistaken for mitral stenosis.[18] One should consider an intracavitary mass when signs and symptoms of tricuspid stenosis are evident, since inflammatory stenosis of this valve is exceedingly rare.[12] Suspected cases of intracavitary cardiac masses may be confirmed by angiocardiogram.

TREATMENT

Operative treatment is the only method available for the removal of this or any other form of cardiac obstruction. Goldberg and associates[5] reported a case of cardiac myxoma diagnosed during life in which an unsuccessful attempt at removal was made. With present day facilities for cardiac surgery, the removal of intracavitary cardiac masses is possible. Once exposed, the removal of such a mass would require but a few minutes. In cases of free ball thrombus the procedure would consist merely in delivering the thrombus. In cases of pedunculated masses, all that would be necessary before delivery would be transection of the pedicle. In the case of a myxoma it remains to be seen whether the entire base must be excised in order to prevent recurrence.

SUMMARY

We have reported the occurrence of an intracavitary cardiac mass diagnosed at necropsy. This lesion could have been removed had it been diagnosed during life. The diagnosis of intracavitary cardiac masses can be made clinically and confirmed by the use of angiocardiograms. With present day facilities for cardiac surgery, the removal of such masses is possible.

An intracavitary cardiac mass should be suspected whenever there is any suggestion of intermittent cardiac obstruction. This obstruction may be pre-

cipitated by the upright position of the patient and may be relieved by the horizontal position. The condition must be thought of in a patient who does not present the usual picture of an ordinary type of heart disease. These patients may show dyspnea that is out of proportion to the physical findings, and digitalization may not give significant relief. The condition should be kept in mind in all patients exhibiting signs and symptoms of marked mitral stenosis. It should be thought of in all patients with tricuspid stenosis, since inflammatory involvement of this valve is rare.

SUMMARIO IN INTERLINGUA

Nos reporta le caso de un thrombo globular intra le cavitate cardiac que esseva diagnosticate al necropsia. Iste lesion haberea essite operabile si illo habeva essite diagnosticate durante le vita del patiente. Le diagnose de massas intra le cavitate cardiac es executabile per medios clinic e pote esser confirmate per le uso de angiocardiogrammas. Con le adjuta del facilitates cardiochirurgic de nostre dies le excision de tal massas es practicabile.

Le presentia de un massa intra le cavitate cardiac deberea esser considerate quandocunque le patiente exhibi signos interpretabile como indicationes de un intermittente obstruction cardiac. Iste obstruction pote esser precipitate per un positura erecte del patiente e se allevia per le positura horizontal. Le presentia de un massa cardiac debe esser prendite in consideration in le casos de patientes qui non exhibi le characteristicas usual del ordinari typo de morbo cardiac. Tal patientes mónstra frequentemente un dyspnea in excesso de lo que esserea a expectar secundo le constatationes physic; e frequentemente digitalisation non resulta in ulle alleviamento significative. Le presentia del condition hic discutite deberea etiam esser suspicite in omne patientes qui exhibi signos e symptomas de marcate stenosis mitral e in illes qui suffre de stenosis tricuspide, proque un affection inflammatori del valvula tricuspide es rar.

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VENTRICULAR ANEURYSM WITH CALCIFICATION OF THE HEART

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ALTHOUGH ventricular aneurysm is not a rare complication of myocardial infarction, its clinical diagnosis during early life is frequently missed. The following case of ventricular aneurysm had the exceedingly rare occurrence of an extensive calcification of the ventricular wall while it presented many of the typical findings described in the literature.

CASE REPORT

History.—This 68-year-old, white male (B.C.) was repeatedly examined at the Cardiac Clinic of Mount Sinai Hospital. He had had gonorrhea in 1910. On June 16, 1946, while working, he suddenly felt a severe, oppressive substernal pain and fainted. He was admitted to Cook County Hospital with the diagnosis of coronary occlusion. He left the hospital, recovered, ten weeks later after 6 weeks of bed rest.

In 1952, the patient was admitted to Mount Sinai Hospital with the diagnosis of coronary occlusion. However, the diagnosis was not confirmed and the patient was dismissed after 1 week.

Occasionally, precordial pain was noticed for about 1 year. Then he resumed his work. In October, 1954, the patient noticed exertional dyspnea and substernal pressure with mild pain transmitted to the left shoulder and arm. Therapy was followed by only a slight improvement.

Physical Examination.—Well-developed and well-nourished man. Slight dyspnea and slight cyanosis of the lips.

Thorax: Evidence of pulmonary emphysema, no moist râles. The breath sounds are diminished, and there is a prolonged expiration.

Heart: There is well visible and extensive systolic pulsation in the third and fourth intercostal spaces at the left of the sternum; this area extends about 3 cm. beyond the midclavian line. A thrust is palpable in the fifth intercostal space. There are no thrills. By percussion, the heart is enlarged to the left with the apex in the fifth intercostal space, 2 cm. beyond the midclavicular line. There is a Grade 2 systolic murmur both at the apex and base, and a Grade 3 systolic murmur at the midprecordium. A triple rhythm is audible at the apex.

Abdomen: The liver edge is palpable one fingerbreadth below the costal margin.

There is no pitting edema of the lower extremities.

The pulse is 60, regular; the blood pressure is 140/80 mm. Hg.

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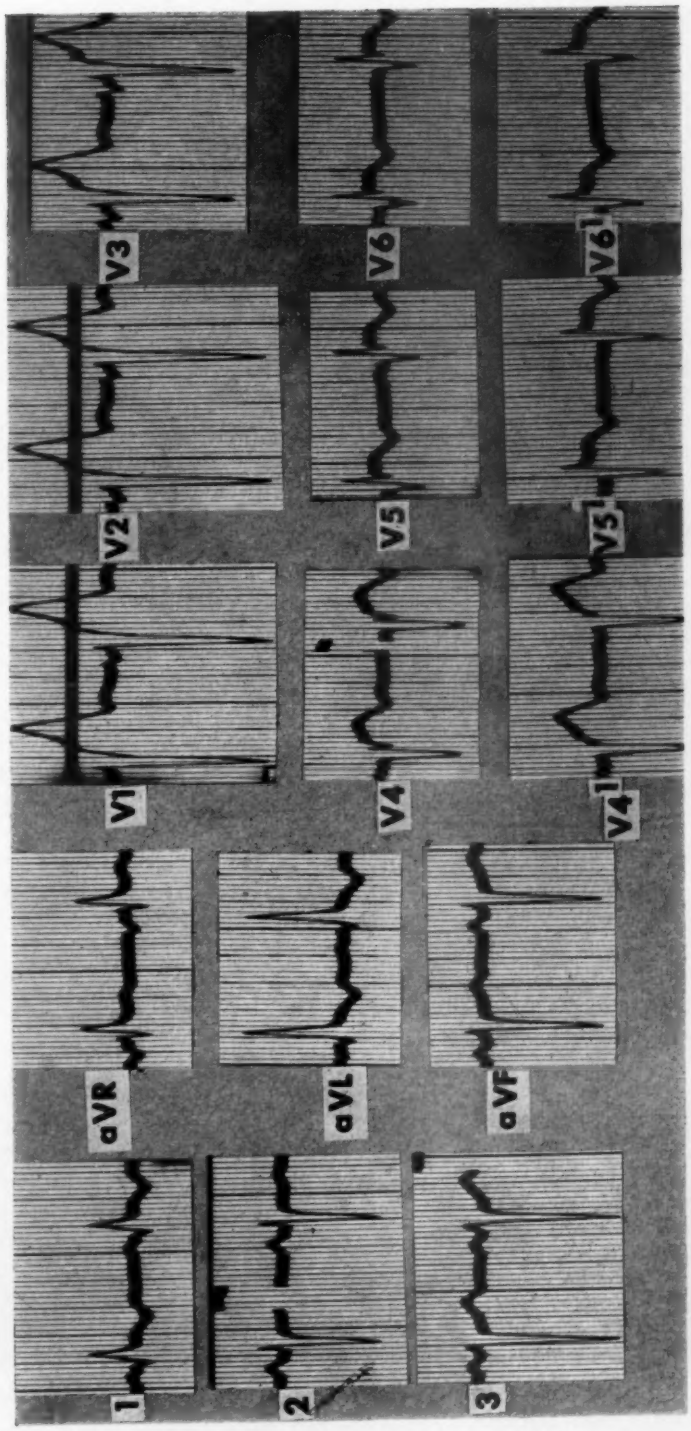

Fig. 1.—Electrocardiogram of Jan. 31, 1955.

Laboratory Examination.—

Electrocardiograms:

Dec. 9, 1950—Sinus rhythm. Slurred ventricular complex in most leads. Duration of QRS = 0.10; R_I, Q_{aVL}, V_6; QS in V_3–V_5. Inverted T_I, T_{aVL}, T_{aVF}, T_{V6}; upright T_{aVR}.

Dec. 21, 1950—Unchanged tracing.

Jan. 11, 1951—More deeply inverted T in I, aV_L, and V_4–V_6.

Jan. 31, 1955—Sinus rhythm. Duration of QRS = 0.14 in the limb leads, 0.16 in the chest leads. QS in V_4, deep Q wave followed by slurring in V_5–V_6 and the high V_5–V_6. Inverted T in I, aV_L, V_5–V_6; upright T in aV_R. Severe elevation of S-T in all the chest leads but particularly in V_4–V_6 and the high V_4–V_6 (Fig. 1).

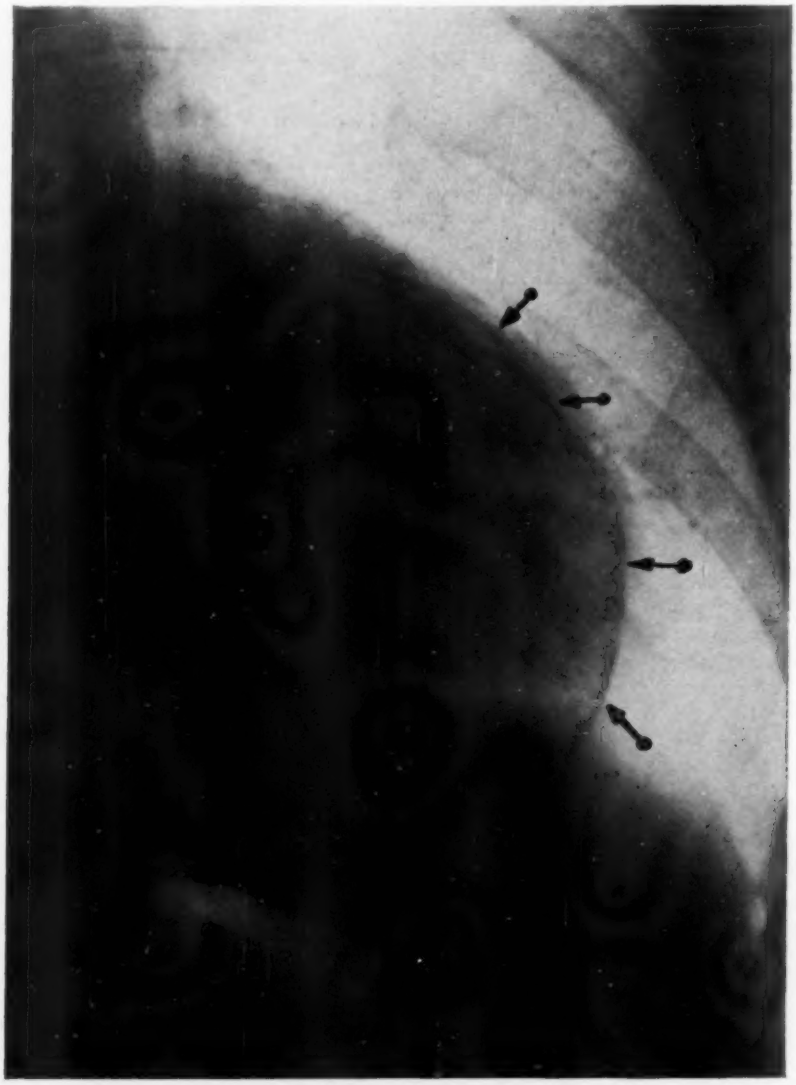

Fig. 2.—Chest film in posteroanterior position.

X-ray: The heart is horizontally placed and has a boot-shaped appearance due to severe enlargement of the left ventricle. In a 30° right anterior oblique (RAO), the upper part of the left ventricle appears bulging and in contact with the chest wall. A thin, dark line, interpreted

as due to calcification, surrounds the lower part of the left ventricle in the posteroanterior (Fig. 2). In RAO, a second line seems to follow endocardial contour of the apex and lower part of the left ventricle (Fig. 3).

Fig. 3.—Chest film in 30° right oblique position.

Phonocardiogram: Low amplitude of first sound at apex. Apical systolic murmur. Prolonged second sound.

Low frequency tracings: Positive pulsations in the third left intercostal space (Fig. 5,*A*); rapid thrust at the apex (Fig. 5,*B*).

Ballistocardiogram: Severely abnormal tracing.

Electrokymogram: Left ventricular tracings recorded in 30° RAO reveal positive pulsations instead of the normal negative waves. In the uppermost part of the ventricle, the pattern of the pulsations has a squarish appearance (plateau-like positive pulsations) (Fig. 4).

DISCUSSION

Nordenfelt[1] and Sigler and Schneider[2] pointed out that the aneurysm of the anterior wall of the left ventricle is accompanied by a low R and a flat or inverted T in Lead I, and by a deep S and a positive T in Leads II and III. While these electrocardiographic changes are not always associated with an aneurysm, a persistent elevation of the RS-T segment should induce the physician to investigate whether a ventricular aneurysm has taken place.[3-6] This

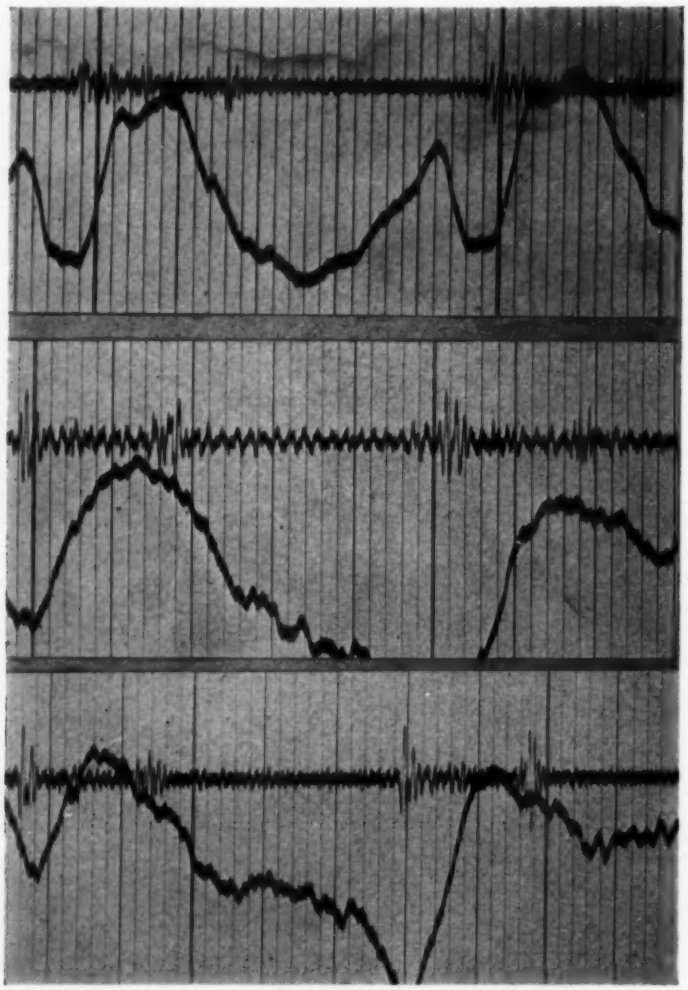

Fig. 4.—Electrokymogram of the anterolateral portion of the left ventricle in a 30° RAO. Three steps from above downward.

elevation has been interpreted as due to ischemia of islands of myocardial fibers within or around the scar tissue.[7] The electrocardiogram of our case is consistent with the previously described pattern. Erroneous diagnosis of fresh infarct was made twice, until attention was called to the persistence of the pattern, and the possibility of a ventricular aneurysm was considered. Certain data of our case indicated left bundle branch block. It is possible that there also was a

peri-infarction block. However, data revealing this cannot be definitely recognized in the presence of bundle branch block.[8,9]

Following the first recognition of aneurysm of the left ventricle by x-ray,[10] several contributions stressed the various diagnostic features of this process. The most significant data, listed by Parkinson and associates[11,12] and by Schwedel and Gross,[13] are the following: (1) enlargement of the left ventricle with deformity of its contour; (2) increase of density of calcification of the ventricular wall; (3) presence of angulations; (4) localized bulge inseparable from the cardiac silhouette; (5) pericardial adhesions; (6) abnormal pulsations.

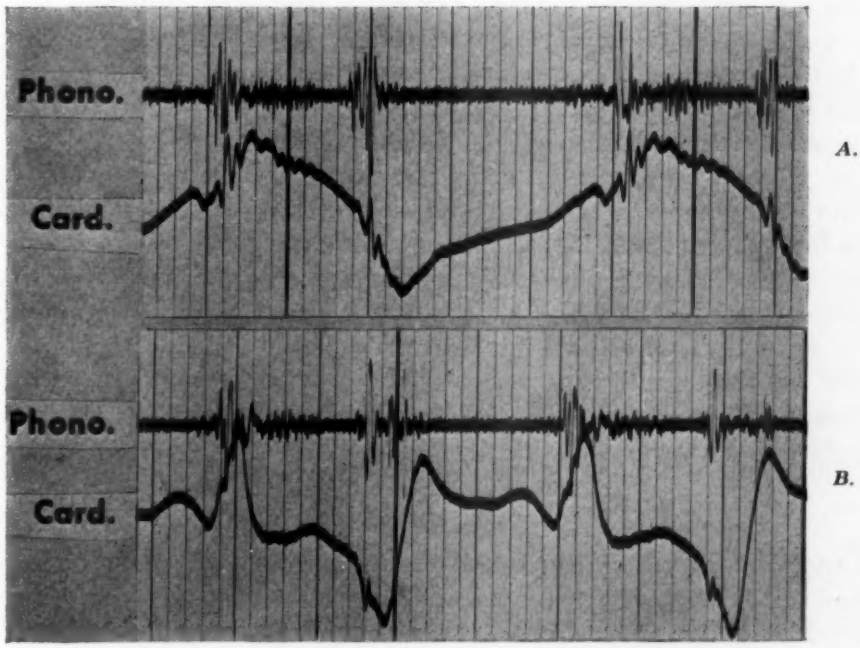

Fig. 5.—Low frequency tracings of the chest wall. Above, sound tracing and expansive pulsation in the third left intercostal space. Below, sound tracing and apex beat in the fourth left intercostal space.

The first description of gross calcification of the wall following a myocardial infarct was published in 1908 by Simmonds.[14] The condition was discovered at autopsy. No additional case was reported until 1924 when Scholz[15] published a case demonstrated by the x-ray during life. Additional reports have subsequently appeared.[16-21]

Calcification of the ventricular wall was recognized during life in rare instances following roentgenologic examination, so that only twenty cases have been reported so far. Diagnosis is important, because of the possible confusion of ventricular calcification with calcific pericarditis and the various prognostic implications inherent in its occurrence.

According to Marks and associates,[22] each calcified plaque of the myocardium is limited outward by a rim of soft tissue of 2 mm. or more which can be observed in all projections. This helps in differentiating the calcifications of the myo-

cardium from those of the pericardium, which are located at the extreme periphery of the cardiac shadow. Movement of the calcified sections with the motion of the heart is in favor of calcification of the myocardium.[22]

The shadow of the calcified area is linear, is limited to the left ventricle, and may be found within the cardiac shadow. That of the calcified pericardium is usually thicker and irregular, and is not limited to the left ventricle.[12]

The x-ray of our case reveals undeniable evidence of calcification of the anterolateral wall of the left ventricle in its low part. All typical roentgenologic data are present.

The electrokymographic pattern of the "dynamic aneurysm" of the left ventricle was described by Luisada and Fleischner[23] and confirmed by others.[24] The term "dynamic aneurysm" was suggested for cases where no definite bulge could be demonstrated in x-ray films while an expansive, positive, systolic pulsation was revealed by electrokymography. A similar type of pulsation was found in three cases of actual aneurysm by Samet and associates.[25]

The electrokymograms of our case reveal a definite expansive positive pulsation in the upper part of the anterolateral wall of the left ventricle. The plateau-like pattern of the highest tracing, similar to a tracing of intraventricular pressure, is an expression of the flabbiness of the wall which expands when pressure increases within it. Less typical patterns are found in lower sections, probably on account of partial calcification of the wall.

The positive systolic thrust revealed by inspection and palpation in the third and fourth left intercostal spaces was definitely caused by contact of the upper part of the aneurysm with the chest wall.

SUMMARY

A case of aneurysm of the anterolateral wall of the left ventricle is reported. The wall of the aneurysm was calcified in its lower part (x-ray demonstration). It expanded during ventricular systole in its upper part causing a typical pattern of paradoxical pulse, proved by electrokymography, and a positive thrust of the chest wall.

SUMMARIO IN INTERLINGUA

Es reportate un caso de aneurysma del pariete anterolateral del ventriculo sinistre. Le pariete del aneurysma esseva calcificate in su parte inferior. Iste facto esseva demonstrate roentgenologicamente. Le aneurysma se extendeva in su parte superior durante le systole ventricular. Assi esseva causate le configuration typic de pulso paradoxe, demonstrate electrokymographicamente. e un impulso positive del pariete thoracic.

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AMAUROSIS FOLLOWING MITRAL COMMISSUROTOMY

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Thomas J. E. O'Neill, M.D., and
Robert P. Glover, M.D.

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MANY reports concerning the results and the postoperative complications following mitral commissurotomy have been written.[1-6] The case described below of amaurosis following mitral commissurotomy is the first in our experience in 750 such operations and the first such complication reported to date.

CASE REPORT

A 34-year-old white male was admitted to the Presbyterian Hospital, Philadelphia, Pa., on Jan. 18, 1955. The pertinent history indicated a rheumatic infection during childhood, otherwise the history was not remarkable. The present hospital admission was due to rapidly progressive pulmonary dysfunction during the past eight months, so that at the time of admission the patient was barely able to walk half a city block. There had been numerous instances of nocturnal dyspnea.

The physical examination revealed a slender, cooperative and seemingly intelligent white male who was not orthopneic or cyanotic. There were no abnormal cervical vessel pulsations. Temperature was 98.2° F., pulse 76/min., respirations 20/min. and the blood pressure 118/80 mm. Hg. The heart was not enlarged to percussion, but there was a prominent apical impulse. The rhythm was regular, 76/min. A mitral diastolic thrill accompanied a Grade 3 mitral diastolic rumble with presystolic accentuation. A Grade 2, early, mitral systolic murmur was audible. M_1 and P_2 were accentuated. The lungs and abdomen were normal. The peripheral vessels of all extremities were accessible and of normal volume, and no abnormal neurologic reflexes were noted.

The laboratory data were as follows: hemoglobin, 13.9 grams; white blood count, 7600, with a normal differential; sedimentation rate 6 mm. in one hour (Westergren). Serology was negative; urinalysis was negative.

An electrocardiogram showed regular sinus rhythm and right ventricular hypertrophy. Fluoroscopy and cardiac x-rays showed moderate enlargement of the left atrium and right ventricle and calcification of the mitral valve.

The patient was considered an excellent surgical risk for operation, and a mitral commissurotomy was done on Jan. 13, 1955. No thrombotic material was detected in the left atrium or appendage. The mitral orifice barely admitted the tip of the index finger of the surgeon (RPG). The cusp margins were heavily beaded with calcium, and the valve leaflets were greatly thickened and little, if any, mobility was felt. The commissures were heavily scarred and the posteromedial one was the site of maximum deposition of calcium. Both commissures were opened

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with considerable difficulty using the guillotine knife, although the resulting enlargement of the mitral orifice was but 1.5 cm. It was speculated at this time that "a piece of calcium might have been dislodged." The procedure was well tolerated, and the operating time was one and one-half hours.

Postoperatively the patient awakened with a left hemiplegia, the signs of which disappeared in the first four hours except for some loss of coordination of the movements of the fingers. But his most persistent and striking complaint was, "I am blind; I can't see anything." The patient seemed rational and cooperative, and at first it was conjectured that a cerebral embolus might be the cause of this symptom. The likelihood of this being true was most improbable in view of a normal ophthalmologic examination, the relatively normal neurologic findings, and the physical condition of the patient. However, the patient continued to complain that he was unable to see; thus, somewhat in desperation a psychiatrist was consulted.

The psychiatric examination on Feb. 18, 1955 revealed the patient to be irritable but only mildly indisposed by his visual deficit. He appeared to be in good contact with his surroundings, and his answers were coherent and relevant. Of singular note was his spontaneously expressed idea that he would be "better off dead." There was little affective correlation of this idea. Questioning evoked a denial of tension, irritability, anxiousness, or nervousness either at present or in the past. He specifically stated that he had not been disturbed in any way by his heart condition or his recent cardiac surgery. His reaction to his amaurosis was one of almost complete indifference. There was evidence in his personality make-up of marked passive dependent traits sharply evidenced by the way he related himself to others and his failure to take responsibility for his own welfare.

Additional history elicited from the family disclosed that the patient had undergone a personality change a few months prior to surgery. He suffered from disturbance of his sleep pattern, had frequent anxiety attacks, and had become more morose and irritable. His intemperate use of alcohol was a major problem in married life and irascibility had increased to such a point that dissolution of marriage appeared imminent.

It was evident that this patient was returning to a state of complete dependence on his mother, and psychiatric hospitalization was recommended. However, this was energetically declined. Worthy of note was the patient's description of visual impressions that he "sees" on the blank wall of his room. These appeared hypnagogic in type.

Recent communications indicate that his cardiac status is excellent, but his visual complaint has persisted unchanged for the past three months. The patient steadfastly avoids and refuses psychiatric treatment.

DISCUSSION AND SUMMARY

It has frequently been pointed out that one of the dread complications of mitral commissurotomy is that of cerebral embolism, and in this instance the transient hemiplegia was most probably caused by dislodgement of a piece of calcium during the procedure. However, there has been no documentation of amaurosis as a complication with or without hemiplegia. In view of the fact that this complication had never presented itself after some 750 operations and the fact that much conjecture and speculation arose as to the possible cause, we felt such a report was worthwhile. It was only after the disappearance of the hemiplegia and the continuation of normal ophthalmologic findings that the patient was considered to have a severe psychic disturbance.

SUMMARIO IN INTERLINGUA

Es presentate un caso de amaurosis occurrente como complication postoperative immediatemente post le execution de commissurotomia mitral. Nulle

altere tal caso se trova in le litteratura. In lor experientias con 750 commissurotomias mitral le autores non ha incontrate ulle caso comparabile.

Le reporto es publicate proque le caso ha provocate multe speculationes e conjecturas in re su possibile etiologia e etiam proque illo esseva accompaniate de transiente hemiplegia sinistre.

Al tempore del presente reporto, i.e. tres menses post le operation, le patiente exhibi un excellente resultato cardiovascular, sed su amaurosis persiste.

Since the submission of this paper for publication another case of amaurosis following mitral commissurotomy has been brought to the attention of the author.

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Book Reviews

EXTRASYSTOLES AND ALLIED ARRHYTHMIAS. By David Scherf, M.D., and Adolf Schott, M.D. New York, 1953, Grune and Stratton, 531 pages, 213 figures, 10 tables. Price $15.00.

This extensive monograph should be in the hands of every cardiologist and, indeed, of all internists who are interested in cardiology.

The book's most engrossing feature is that dealing with the history of the development of our knowledge of these arrhythmias where due credit is given to the pioneers who blazed the trail for all who followed.

It is but natural that the statement of present-day concepts should be based largely upon the personal work of the authors in both the experimental and the clinical fields. Their experience covered such a wide range that it would be most difficult to review all aspects of the monograph in detail. There are some statements and interpretations that might not be agreed to by certain readers but, after all, within our present-day knowledge these could be regarded as matters of opinion. It is such differences that offer a challenge to further investigations into the various subjects outlined in the monograph. It must be appreciated that cardiology is not yet a static science; it still requires a dynamic approach to its many problems. Throughout the whole book there is such an approach and, although the authors express their own interpretations, these are not, in any fashion, so dogmatic as to close the door. Rather, by inference, they suggest further avenues for exploration.

The monograph, therefore, should appeal not only to the practicing cardiologists, but also to those interested in fundamental research.

S.C.M.

BLUTGERINNUNGSFAKTOREN. By Erwin Deutsch, Vienna, 1955, Franz Deuticke, 298 pages, 35 figures, 32 tables.

This is a comprehensive and up-to-date presentation of the biochemistry of blood coagulation. Details of clinical problems are omitted but numerous references on the changes of fundamental factors in various diseases are given. Dicumarol effects are quite extensively treated.

The introduction gives a short outline of the main phases of blood coagulation and interactions of the various factors, illustrated by a two-page diagram, which facilitates the understanding of the detailed discussion in the following chapters. Since many of the problems are controversial now, the author gives major attention to what he considers to be the best current hypothesis, but also opposing views are discussed.

The book follows the time sequence of blood coagulation: The Preliminary Phase (formation of active thrombokinase) is discussed in Chapter II (pp. 7-62), the First Phase (activation of prothrombin to thrombin) in Chapter III (pp. 63-133), the Second Phase (formation of fibrin from fibrinogen) in Chapter IV (pp. 133-164), the Third Phase (retraction) in Chapter V (pp. 166-172), and the Fourth Phase (fibrinolysis, which may or may not occur) in Chapter VI (pp. 173-198). Chapter VII discusses the effect of other enzymes (trypsin, snake venoms, staphylocoagulase, etc.) on blood coagulation.

After the reader has worked through the details of one of the most intricate biological reactions, he will welcome the brief synopsis in the final chapter, which also contains an extremely useful tabulation of the synonyms used by the various authors (pp. 227-229).

E. S.

KREISLAUFREGULATION. By H. Reindell, E. Schildge, H. Klepzig, and H. W. Kirchhoff, Stuttgart, 1955, Georg Thieme Verlog, 315 pages, 19 tables, 65 figures.

Although the physiological basis of circulatory regulations (Chapter II, pp. 3-86) and their disturbance in various cardiovascular disorders (Chapter V, pp. 111-161) are discussed in some detail, the main emphasis of the book is placed on the psychosomatic aspects of cardiovascular disease. The attempt to explore the possible mechanisms of psychosomatic interaction with the purpose "of a better understanding of the phenomena observed in patients with cardiovascular disease" (p. 2) is certainly a worthwhile undertaking. Most of the clinical material is taken from the file of the senior author, including case histories, clinical findings, and laboratory tests (stroke volume, peripheral resistance, ECG in rest, standing, and after exercise, etc.).

Hypnosis experiments were performed in order to explore the various types of interference of emotional effects with circulatory functions, and their significance for the production of circulatory disorders. In some patients with a psychic trauma, reproduction of the trauma in hypnosis elicited arrhythmias from single extrasystoles to ventricular tachycardia (Fig. 41, p. 215), significant S-T depression or T-wave inversion, together with hemodynamic changes, which, in one patient, included also an increase of pressure in the pulmonary artery. The authors are aware, however, that the psychosomatic relationship is extremely variable, not only from patient to patient, but also within the same patient. The demonstration of significant hemodynamic and ECG changes under emotional stress raises the question of diagnostic differentiation between nervous and organic circulatory disorders. The problem appears to be, if anything, more difficult than before; the authors maintain that long-lasting emotional stress may ultimately produce permanent cardiovascular disorders in patients with "a somatic type of reaction." Therefore, there seems to be a fluid transition between nervous and organic circulatory disturbance rather than a sharp differentiation.

The attempt of a synthesis is handicapped by the fact that there is still a wide gap between the safely established experimental information and the very complex phenomena observed in patients. Therefore, much of the discussion is speculative and not always critical. The authors accept the common stress hypothesis of the evils of modern civilization, but do not consider the contradiction implied by the drop in the incidence and mortality of cardiovascular disease in many European countries during the much greater stress of war. In the preface by Heilmeyer it is stated that any emotional stress affects the heart of man of Western culture (uns abendländischen Menschen) implying that this may be not the case for the Oriental man, and this thought is repeated later (p. 253). No attempt is made to support these statements by more objective evidence of absence of emotional stress effects in other than Western cultures.

The last two chapters are concerned with prognosis and therapy (pp. 254-309).

As a whole, interesting material is presented for thought in an area which merits increasing consideration, and in this respect the book serves the useful purpose the authors had in mind.

E. S.

CARDIAC EMERGENCIES AND HEART FAILURE: PREVENTION AND TREATMENT. By Arthur M Master, Marvin Moser, and Harry L. Jaffe, Ed. 2, New York, 1955, Lea & Febiger, 203 pages.

In this second edition, the authors, who are well known cardiologists, have been able to include important advances in treatment without increasing the size of the book greatly. This is not just a revision with addition of new material. The book has been completely rewritten from cover to cover and is therefore up-to-date. The important aspects of treatment are covered in sufficient detail so that the doctor can really use this book for ready reference in an emergency.

The serious arrhythmias, acute heart failure, coronary artery disease and its complications, hypertensive crises, and cardiac complications arising during surgical treatment are some of the subjects covered. There are a few well chosen illustrations and an excellent bibliography of over 300 references. The index is good, as it should be in a book of this kind. The interne or resident physician can slip this small volume into his pocket to have it available for any acute cardiac situation on his service. The book is also valuable for the general practitioner.

A. D.

HEART DISEASE (ITS DIAGNOSIS AND TREATMENT).—By Emanuel Goldberger, M.D., F.A.C.P., Ed. 2, Philadelphia, 1955, Lea and Febiger, 781 pages. Price $12.50.

This practical text has been enlarged by 130 pages to include the advances of cardiology in recent years. It is divided into five sections.

Section I.—The Normal Heart proceeds directly to the description of normal physical findings and the special recording methods. A new chapter has been added on ballistocardiographic examination. *Section II.*—The Abnormal Heart begins with symptoms, abnormal physical findings or signs from radiology, electrocardiography, and ballistocardiography, and a circulatory efficiency test. *Section III.*—Cardiac Syndromes begins with congestive heart failure and the newer concepts of mechanisms and treatment with reference to electrolyte disturbances are discussed. The shock syndrome, syncope, neurocirculatory asthenia, angina pectoris and cardiac arrhythmias are discussed. *Section IV.*—Systematic Description of Cardiac Abnormalities begins with the congenital heart disease section which has been expanded and includes method of diagnosis and descriptions of cardiac catheterization, and oximetry and dilution curves have been added. Rheumatic fever, collagen diseases, bacterial endocarditis, and some material on newer antibiotics, contraindications for mitral valvulotomy have been appended. Arteriosclerotic coronary artery disease, myocardial infarction, and hypertension chapters have added material. The use of the newer sympathetic blocking, antihypertensive drugs, have been newly outlined but, unfortunately, Ansolysen is not mentioned. The chapters on pulmonary hypertension, pulmonary heart disease or cor pulmonale have been rewritten and the newer concepts of its treatment have been added. There are also chapters on the rarer types of heart disease. *Section V.*—Special Conditions Complicating Heart Disease, and some new material on Work Classification of Cardiacs is added.

The therapeutic directions are sound and helpful and emphasize the quite extensive practical experience of the author. The student, general practitioner, and internist can study this book to advantage and profit considerably from it. There are 298 illustrations, 107 figures, and 5 tables.

S.R.H.

CARDIOLOGY NOTEBOOK. By Fishman et al., Columbia University College of Physicians and Surgeons, New York, 1955, Grune and Stratton, Inc. Price $2.50.

Cardiology Notebook, by a group of distinguished authors, is an authentic, highly simplified presentation of the elements of (1) cardiovascular fluoroscopy, (2) electrocardiography, (3) hemodynamics, and (4) nomenclature for cardiac diagnosis.

It should be in the library of every medical student beginning his clinical years.

A.J.M.

BALISTOCARDIOGRAFIA CLINICA. By Sergio Alvarez, Havana, Cuba, 1955, Manuel V. Fresnada, 333 pages with illustrations.

This book constitutes a review of the literature in ballistocardiography from the middle 1930's to early 1954. Most of the significant papers published in that period have been quoted. Though it provides a useful review, it does not present a critical evaluation of methods or results. The style makes reading easy even though the reader's knowledge of Spanish may be limited.

J.N.

ELECTROCHEMISTRY IN BIOLOGY AND MEDICINE. Edited by Theodore Shedlovsky, Sponsored by The Electrochemical Society, Inc., New York, 1955, John Wiley and Sons, Inc.

This book presents the report of a symposium on electrochemistry in biology and medicine held in New York in April, 1953. Having numerous authors it has an expected variety of styles and topics. The tendency of the writers to stress subjects of particularly current interest makes the book of less interest to a general reader than to the specialist in the field to whom the book provides a valuable reference text.

J.N.

Announcement

THE COMMITTEE ON CARDIOVASCULAR DISEASE OF THE COUNCIL ON RESEARCH, AMERICAN COLLEGE OF CHEST PHYSICIANS, offers an award of $500 for the best manuscript on ACUTE PULMONARY EDEMA. The study may be of either an experimental or a clinical type and may include problems of therapy. The original work, based on personal research, should be presented before May 1, 1956. It may consist of an unpublished manuscript or a recently published article (after April 1, 1955).

If the manuscript is unpublished, publication may take place either in *Diseases of the Chest* or in another journal, according to the wish of the author.

For further information, please communicate with Dr. Aldo A. Luisada, Chairman, Section on Cardiovascular Physiology, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois.

Erratum

In the August, 1955, issue of the JOURNAL, in the article by Likoff, Berkowitz, Geyer, Strauss, and Reale entitled "Plasma, Red Cell, and Total Blood Volume Changes Following Cardiac Surgery," on page 164 in the first sentence under "Results," the mean net change in plasma volume should read "+5.2 c.c. per kilogram" instead of "−5.2 c.c. per kilogram."

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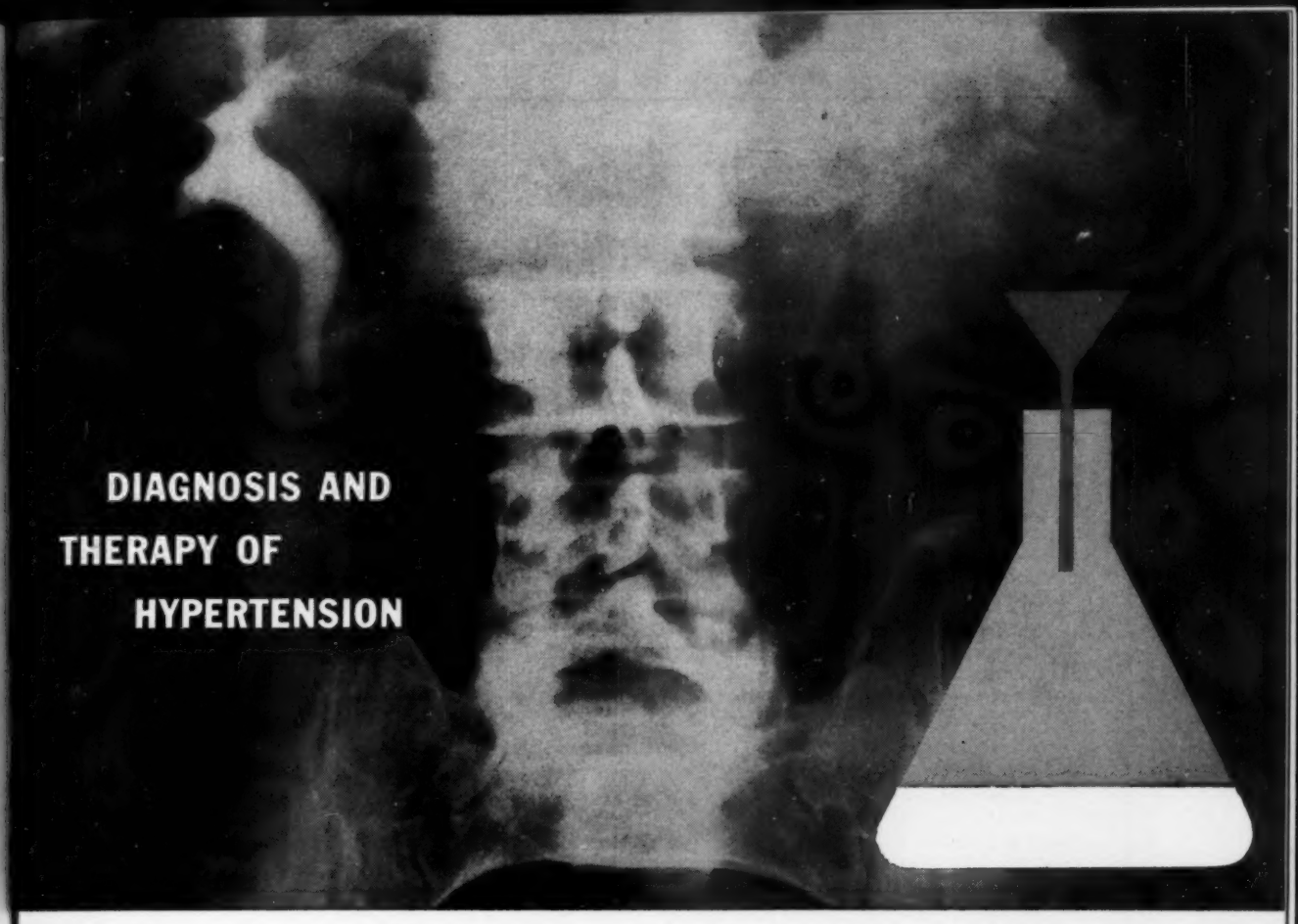

kidney damage

In the diagnosis of hypertension, kidney damage can be recognized by *two* commonly used methods: blood chemistry may indicate functional inadequacy; the retrograde pyelogram reveals structural damage.

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*Dennis, E.; McConn, R. G.; Ford, R. V.; Hughes, W. M.; Beazley, H. L., and Moyer, J. H.: Postgrad. Med. *16*:300 (Oct.) 1954.

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JONATHAN C. MEAKINS, M.D., Editor

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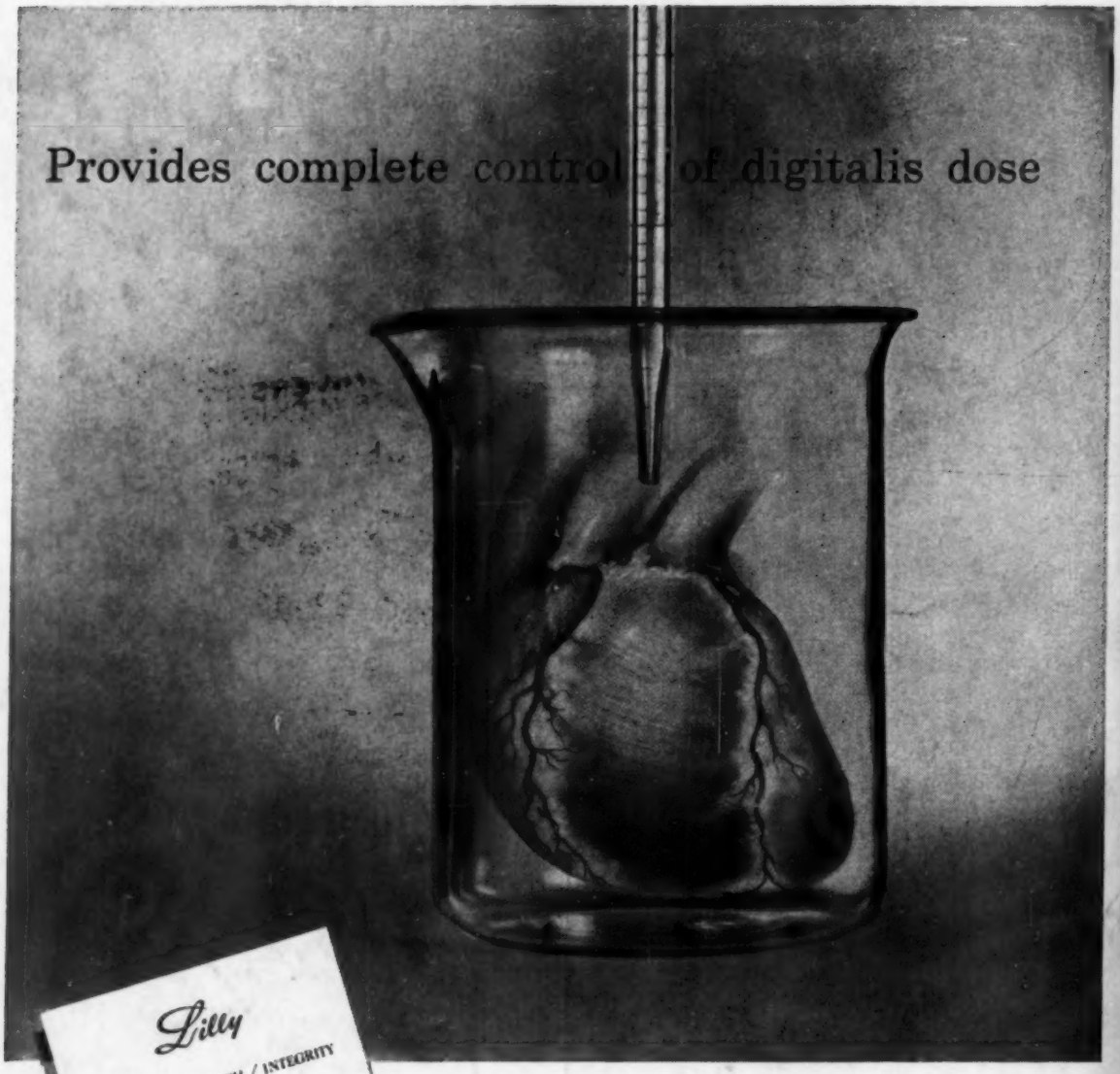

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